Heart Failure Essentials for Cardiology Fellows 2016

Management of Acute Heart Failure

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Ramathibodi hospital

Slide

Powervote Setting

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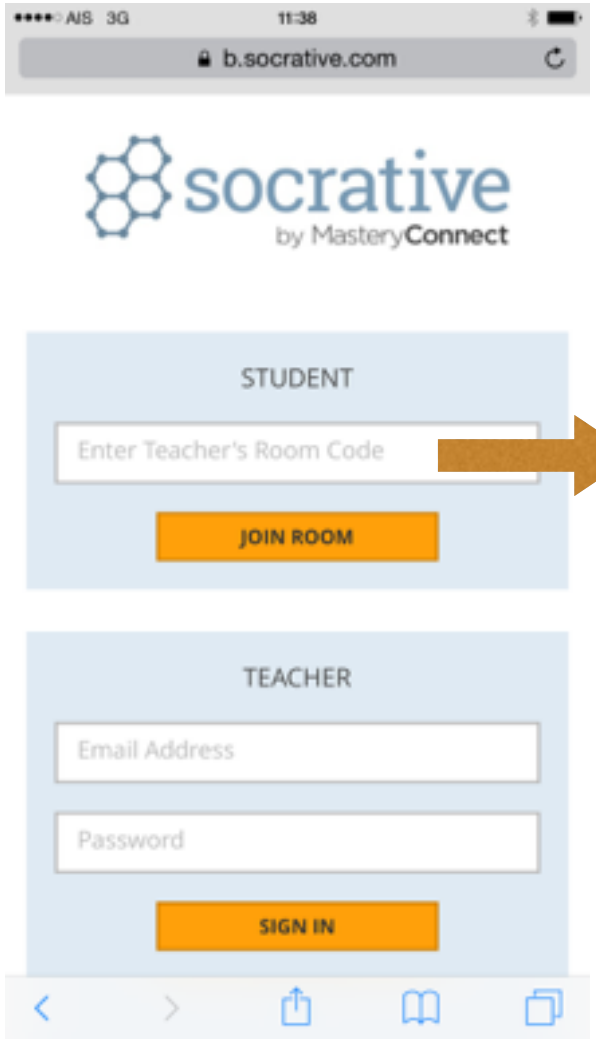
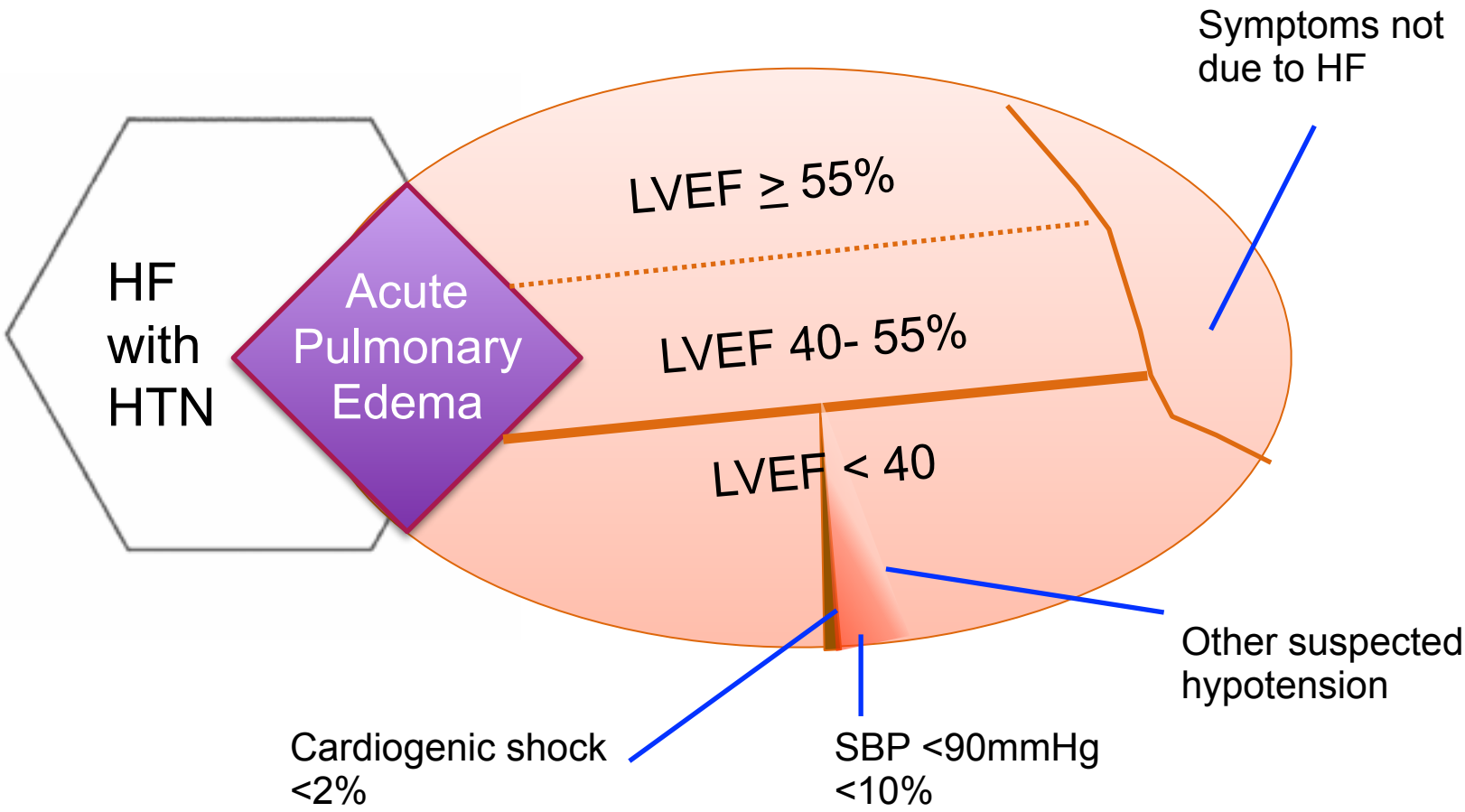


TEERAPAT

กรอกชื่อ

Typical proportions of HF hospitalization in ADHERE registry



J Am Coll Cardiol, 47,76-84

Case #1

64-year-old female

NICM EF 25%, returned from vacation 2 days ago

DOE, walking distance ↓ from 1 km to 150 m

4-pillow orthopnea, 4 kgs weight gain

PE: alert and oriented, BP 105/75 mmHg, P=82
JVP 15 cmH_2O, bibasilar rales, 2+ edema, warm extremities

Cr 1.6 (baseline 1.2)

On Carvedilol, Lisinopril, Spironolactone, Furosemide

What is the best initial therapy

A. Stop beta blocker and ACE I

B. Start IV furosemide at 1- 2.5 times of the home oral equivalent dose

C. Start Dobutamine drip

D. Start Milrinone drip

E. Instruct the patient not to take anymore vacation

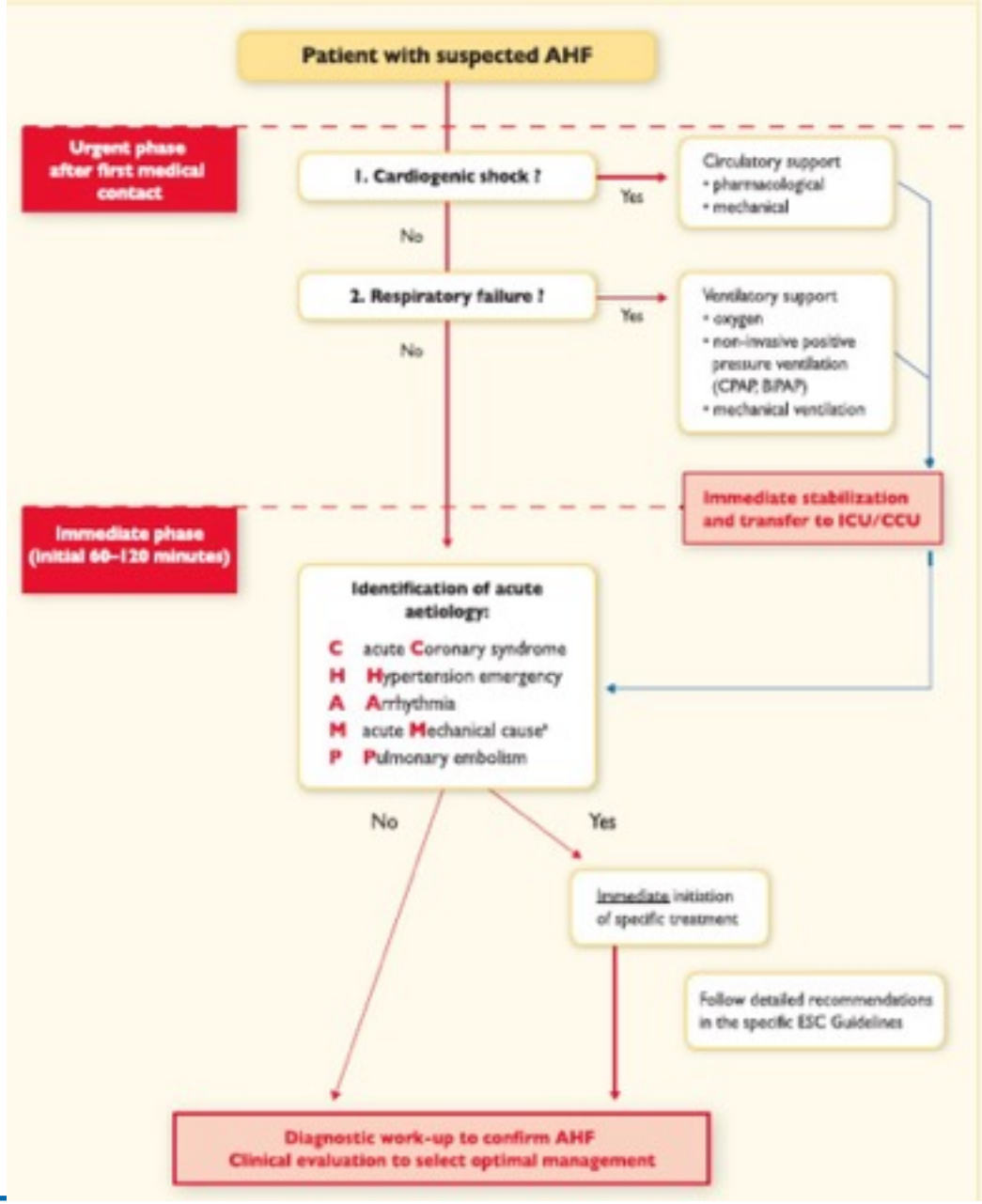
European Heart Journal
doi:10.1093/eurheartj/ehw128

ESC GUIDELINES

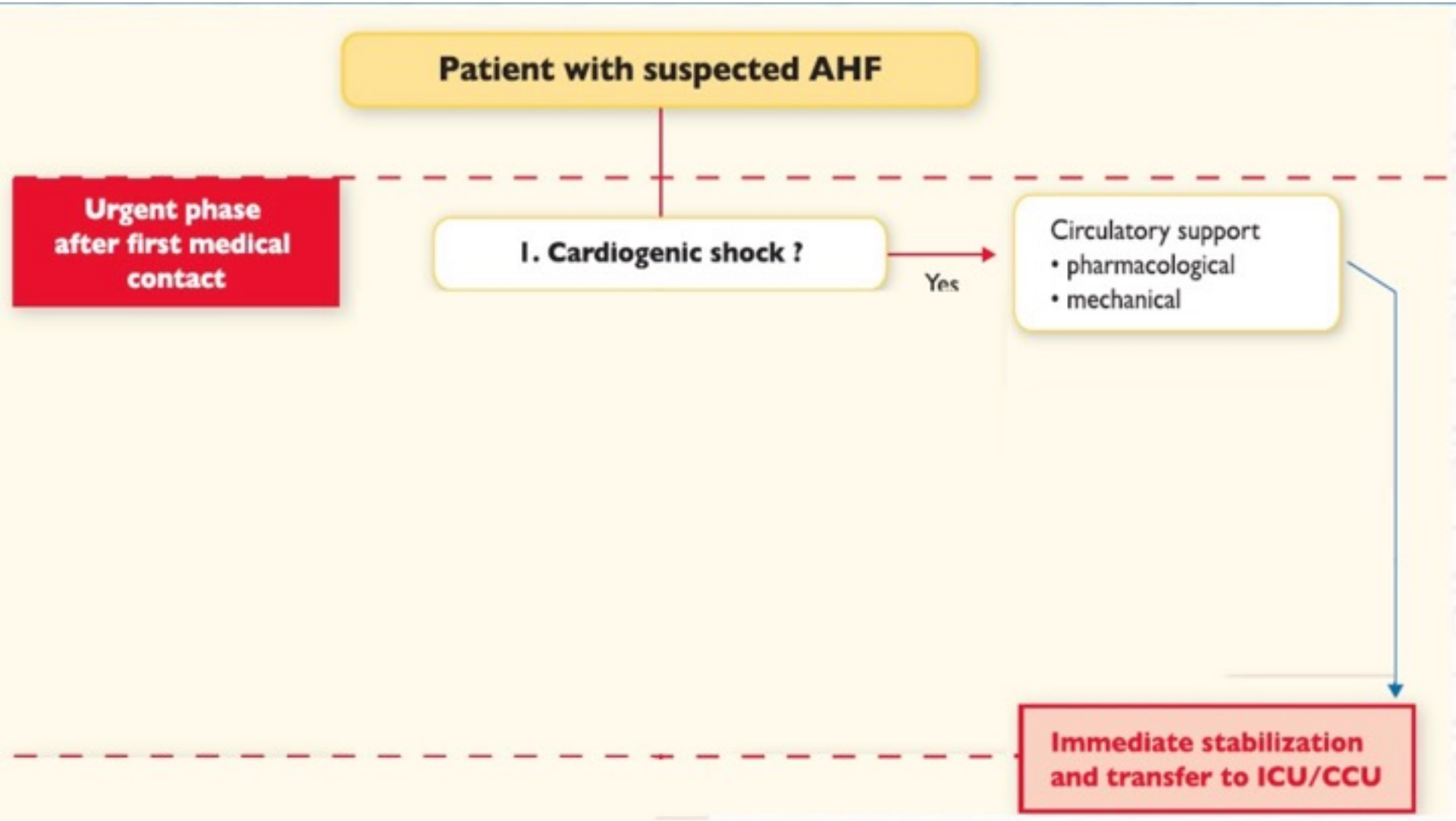
2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure

The Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC)

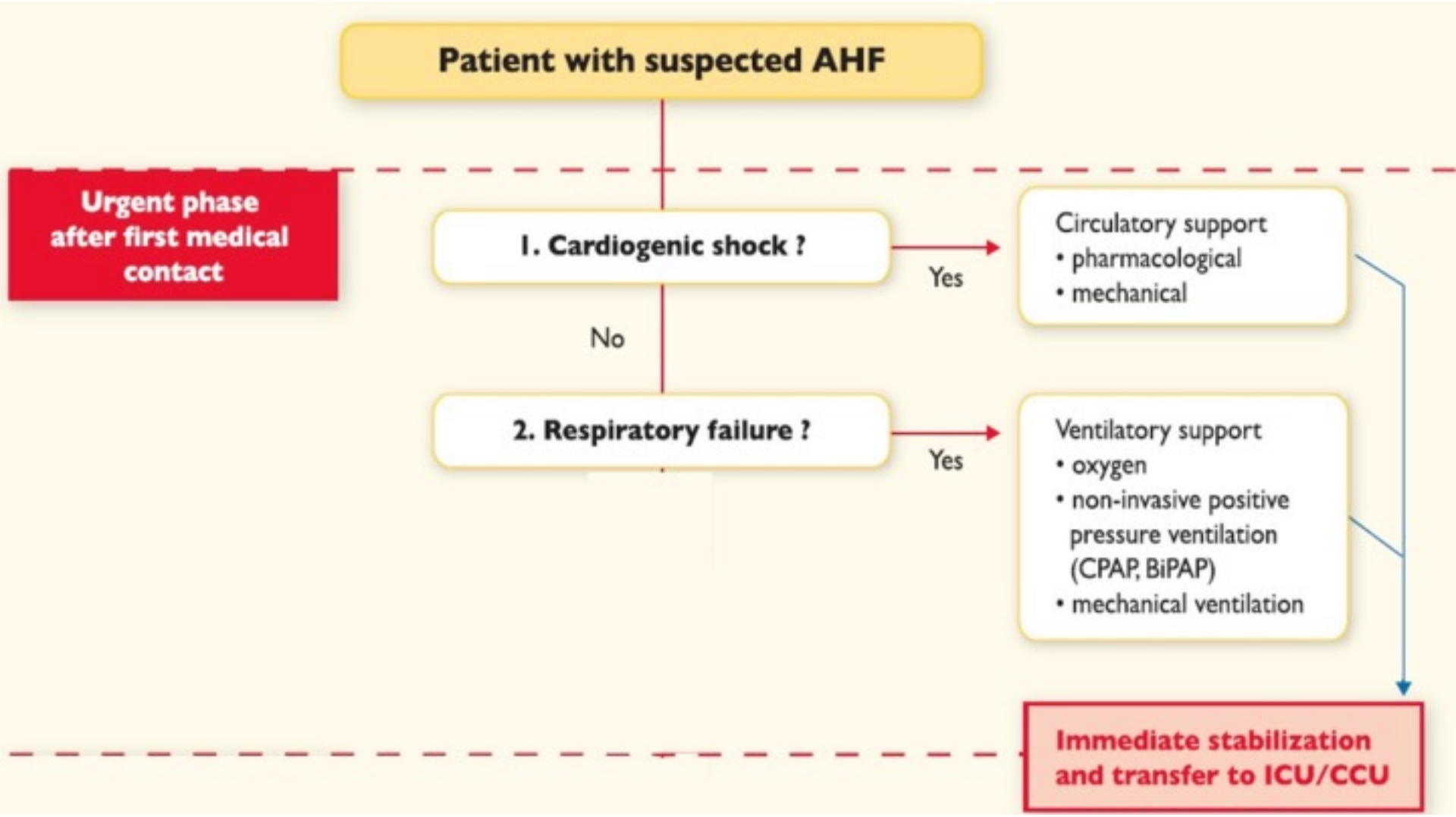
Developed with the special contribution of the Heart Failure Association (HFA) of the ESC


Patient with suspected AHF
Urgent phase after first medical contact
1. Cardiogenic shock ?
Yes
Circulatory support
• pharmacological
• mechanical
No
2. Respiratory failure ?
Yes
Ventilatory support
• oxygen
• non-invasive positive pressure ventilation (CPAP, BiPAP)
• mechanical ventilation
No
Immediate stabilization and transfer to ICU/CCU
Immediate phase (initial 60–120 minutes)
Identification of acute aetiology:
C acute Coronary syndrome
H Hypertension emergency
A Arrhythmia
M acute Mechanical cause*
P Pulmonary embolism
No
Yes
Immediate initiation of specific treatment
Follow detailed recommendations in the specific ESC Guidelines
Diagnostic work-up to confirm AHF
Clinical evaluation to select optimal management

Initial Management of AHF



Initial Management of AHF



Oxygen and ventilation Rx

Monitoring of transcutaneous arterial oxygen saturation (SpO_2) is recommended

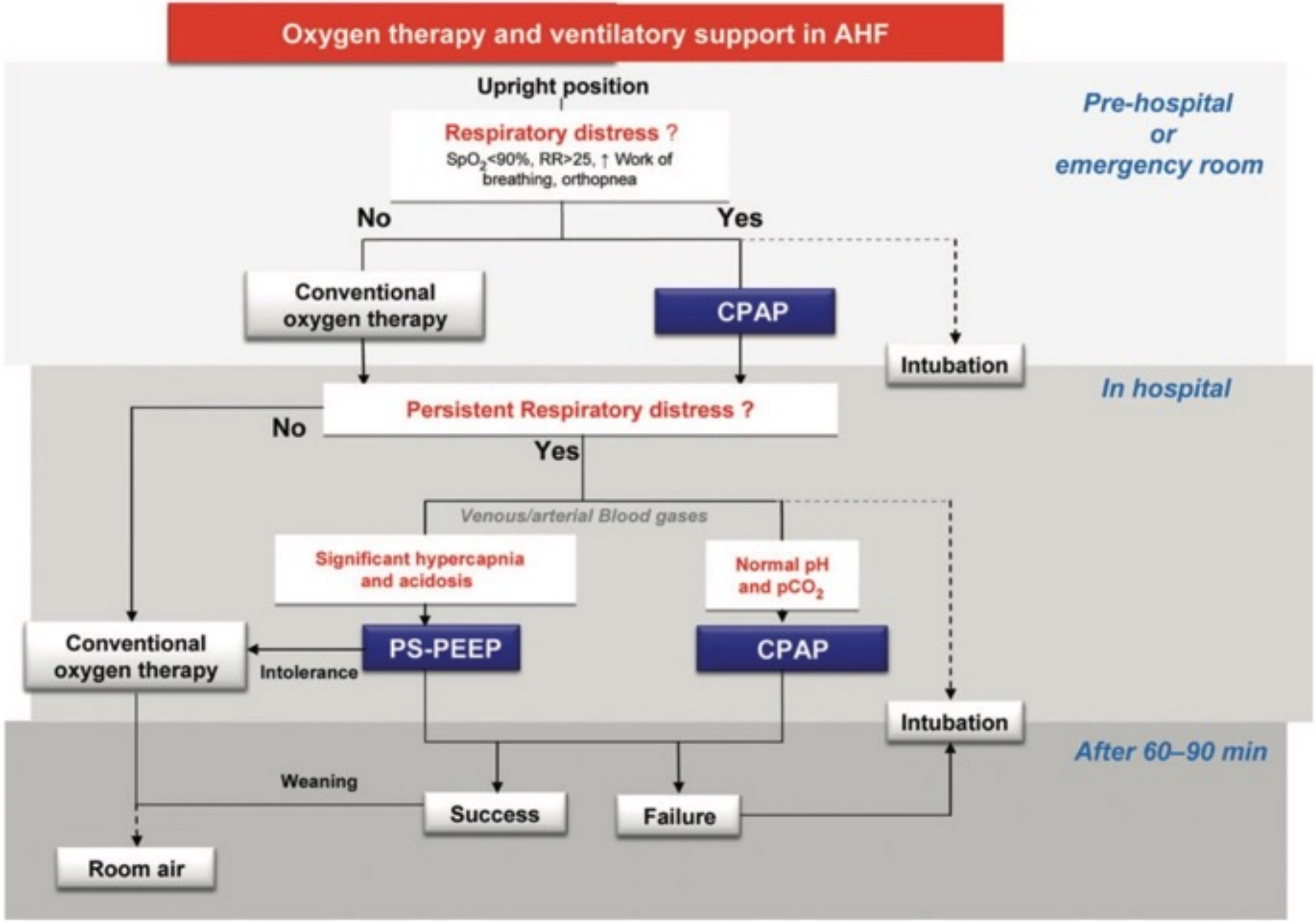
Oxygen therapy is recommended in patients with AHF and SpO_2 <90% or PaO_2 < 60 mmHg to correct hypoxemia

Intubation is recommended, if respiratory failure,leading to hypoxemia (PaO_2 < 60), hypercapnia ($PaCO_2$>50 mmHg) and acidosis (pH<7.35), cannot be managed non-invasively

Oxygen and ventilation Rx

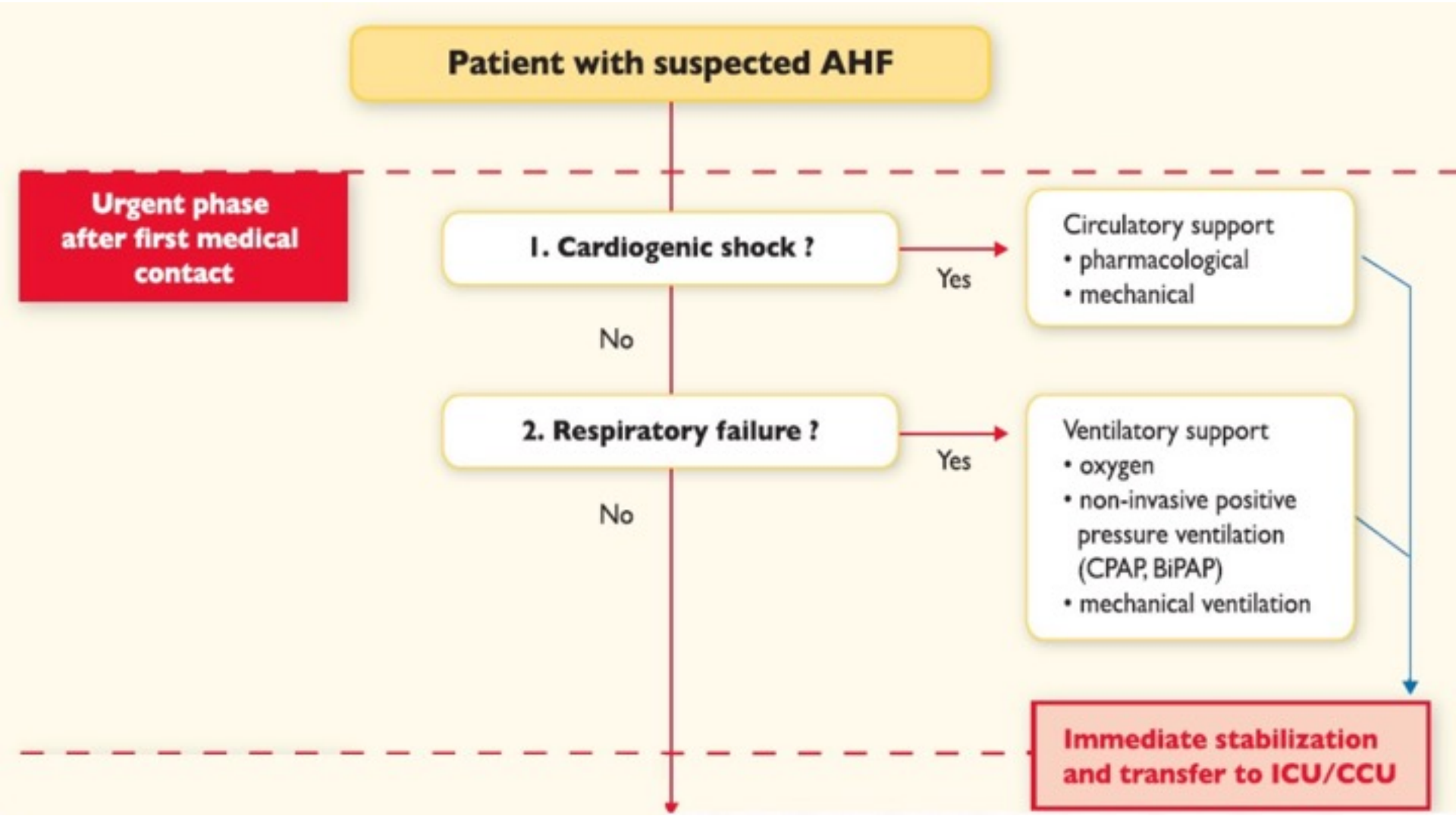
Non-invasive positive pressure ventilation (CPAP, BiPAP) should be considered in patients with respiratory distress (RR>25/min, SpO2 <90%) and started as soon as possible in order to decrease respiratory distress and reduce the rate of mechanical endotracheal intubation

Non-invasive positive pressure ventilation can reduce blood pressure and should be used with caution in hypotensive patients. Blood pressure should be monitored regularly when this treatment is used.



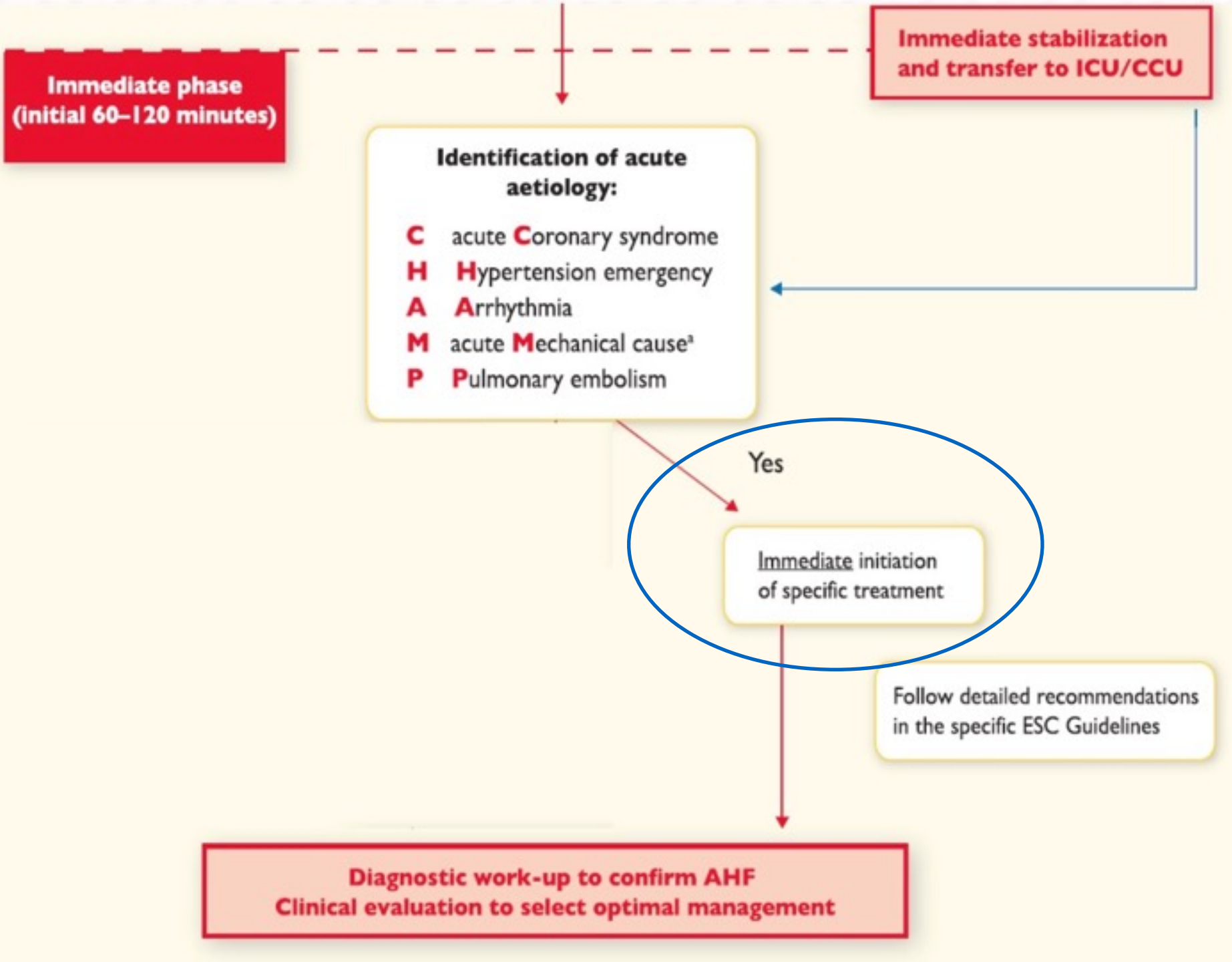
European Journal of Heart Failure (2015) 17, 544–558

Initial Management of AHF



Criteria for ICU/CCU admission

- Hight risk patients (persistent significant dyspnea, hemodynamic instability, severe arrhythmias, AHF due to ACS)
- Need for intubation (or already intubated)
- Signs/symptoms of hypotension
- SpO2 < 90% despite supplemental oxygen
- Use of accessory muscles for breathing, RR>25/min
- Heart rate <40 or >130 bpm, SBP < 90 mmHg


Immediate phase
(initial 60–120 minutes)
Immediate stabilization and transfer to ICU/CCU
Identification of acute aetiology:
C acute Coronary syndrome
H Hypertension emergency
A Arrhythmia
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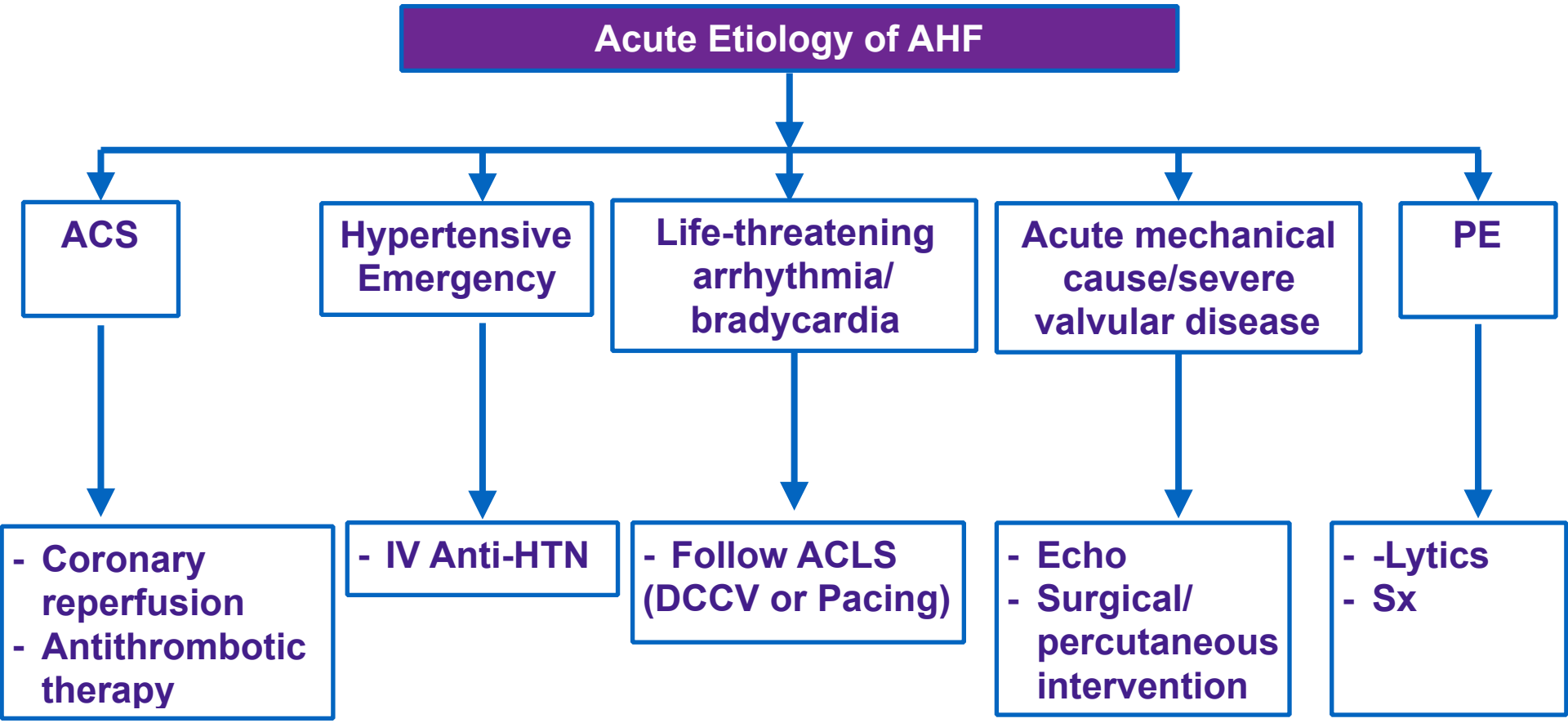
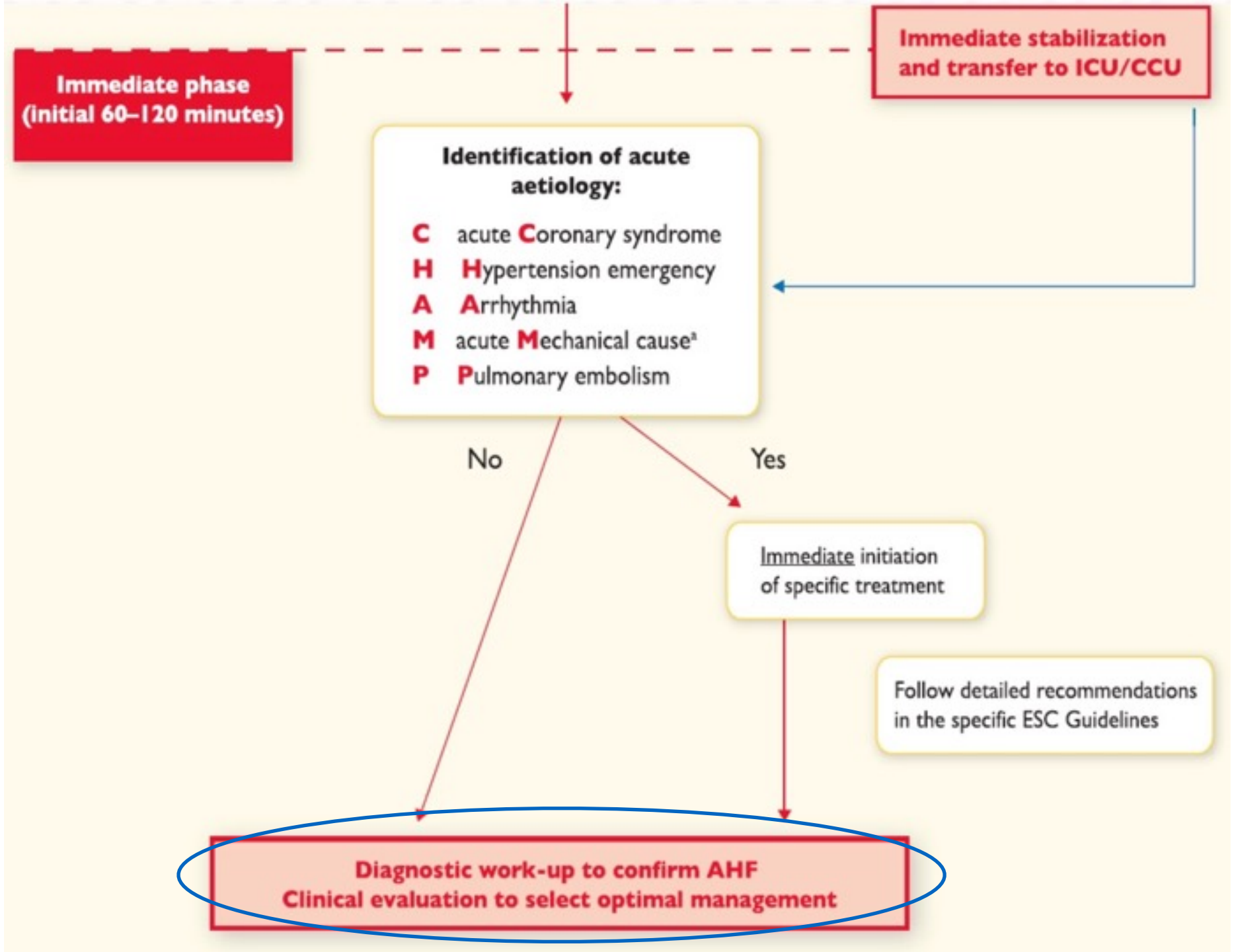
P Pulmonary embolism
Yes
Immediate initiation of specific treatment
Follow detailed recommendations in the specific ESC Guidelines
Diagnostic work-up to confirm AHF
Clinical evaluation to select optimal management


Acute Etiology of AHF
ACS
Hypertensive Emergency
Life-threatening arrhythmia/ bradycardia
Acute mechanical cause/severe valvular disease
PE
- Coronary reperfusion
- Antithrombotic therapy
- IV Anti-HTN
- Follow ACLS (DCCV or Pacing)
- Echo
- Surgical/ percutaneous intervention
- -Lytics
- Sx


Immediate phase
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Yes
Immediate initiation
of specific treatment
Follow detailed recommendations
in the specific ESC Guidelines
Diagnostic work-up to confirm AHF
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Diagnosis and initial prognostic evaluation

Lab test at presentation

Natriuretic peptides

Acute heart failure is unlikely if :

BNP < 100 pg/mL (vs 35 pg/mL in chronic setting)

NT-proBNP < 300 pg/mL (vs 125 pg/mL in chronic)

MR-proANP < 120 pg/mL

Other labs

cTn,BUN, Cr, Electrolytes, LFT, TSH

European Journal of Heart Failure (2015) 17, 544–558

Diagnosis and initial prognostic evaluation

Additional investigations

ECG

- Underlying cardiac diseases (AF, ischemia)
- Rarely normal in AHF

CXR

Normal in up to 20% of AHF

Echo

Preferably within 48 hours from admission

Immediate : Cardiogenic shock or life threatening structural CV diseases

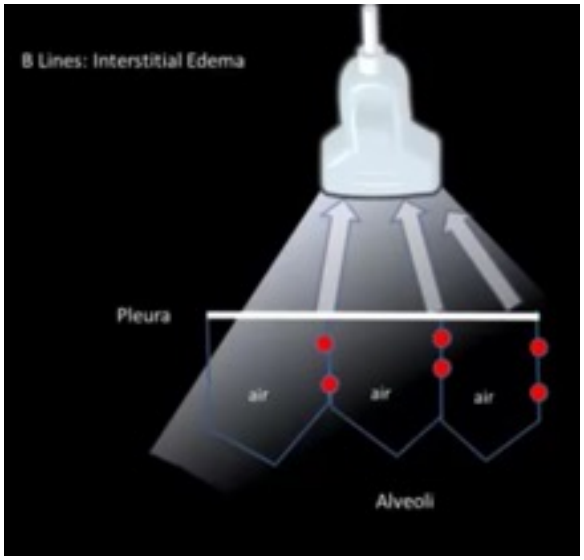
European Journal of Heart Failure (2015) 17, 544–558

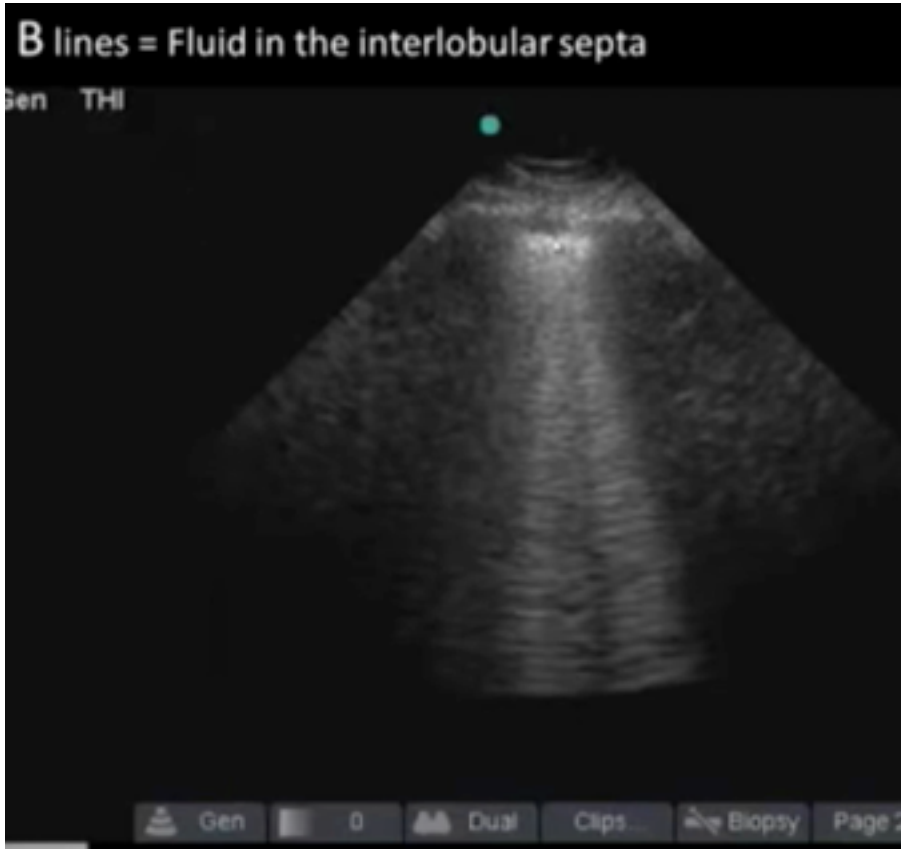
Diagnosis and initial prognostic evaluation

Lung ultrasounds

B Lines

- Vertical, hyper echoic rays projection from pleural line (ring down artifact)
- Reflects fluid in the interlobular septum





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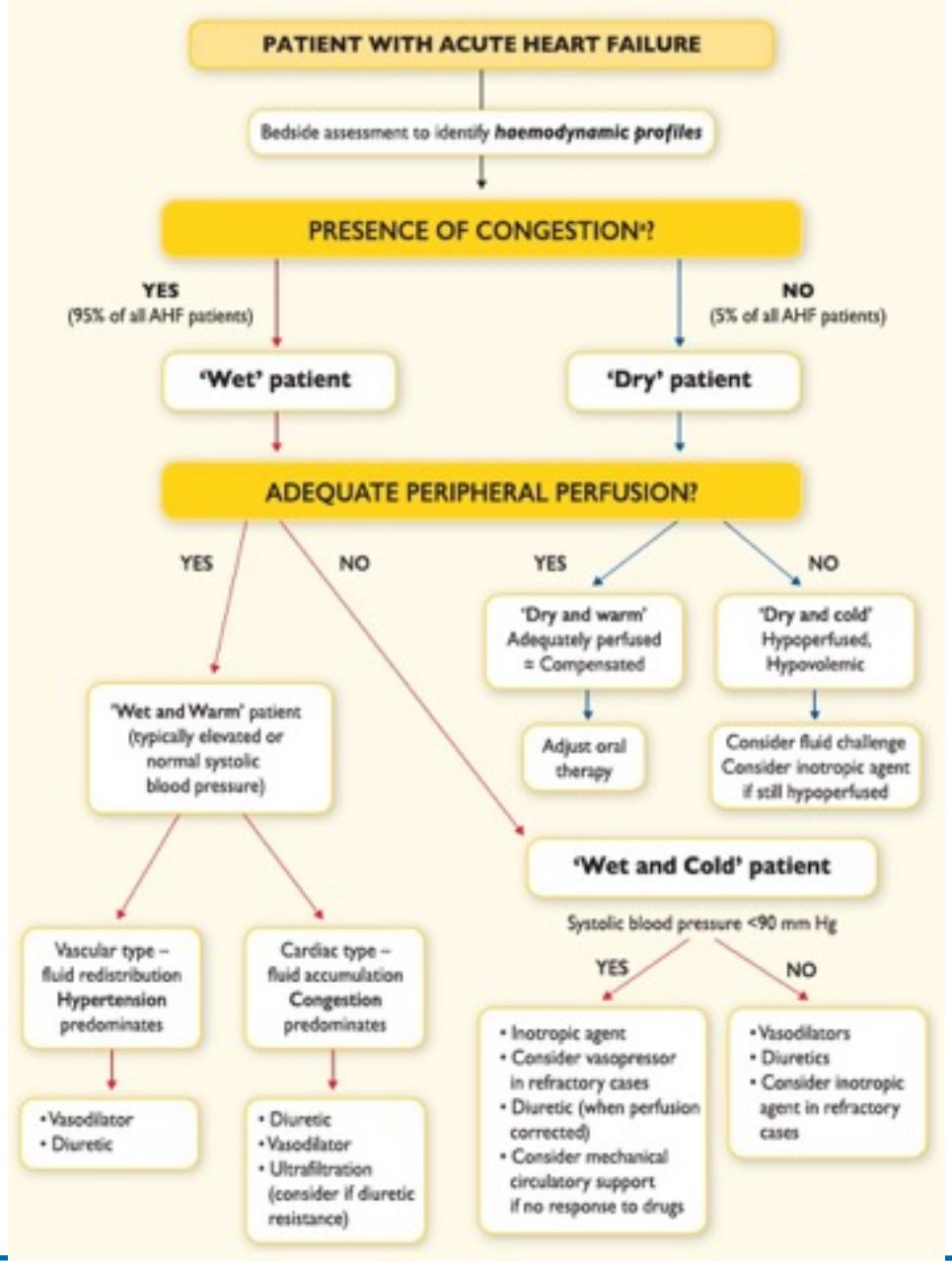
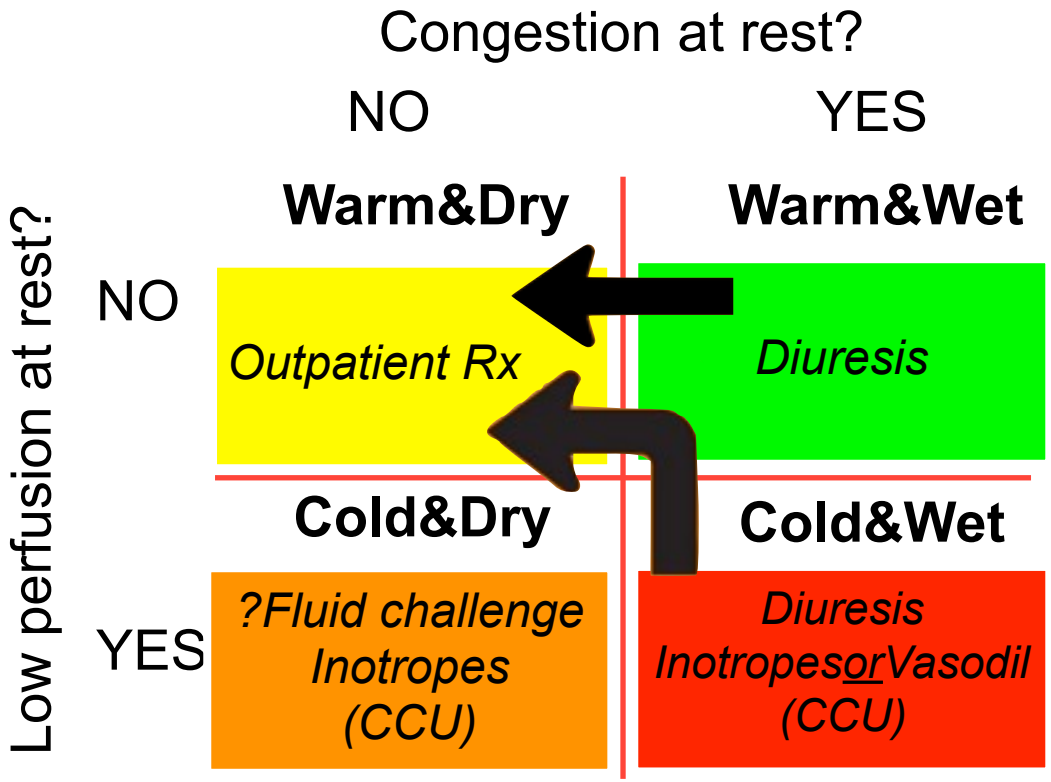


Eu Heart J May 20, 2016

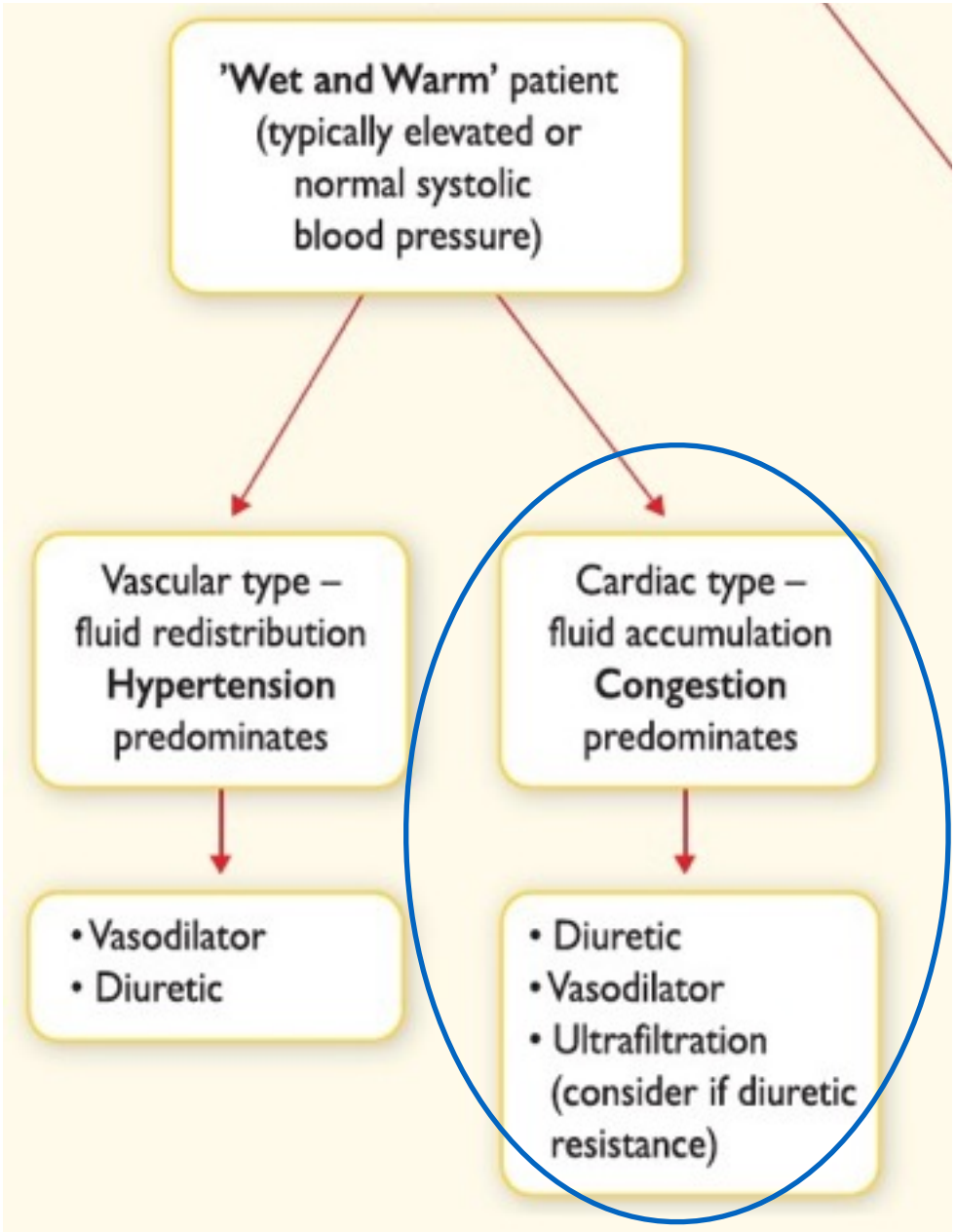
PATIENT WITH ACUTE HEART FAILURE

Bedside assessment to identify ***haemodynamic profiles***



Evidence for congestion
- Orthopnea/PND
- Jugular venous distention
- Peripheral (bilateral edema)
- Congested hepatomegaly
- Gut congestion, ascites
- Hepatojugular reflux
- Valsalva square wave

Evidence for low perfusion
- Cold sweated extremities
- Oliguria
- Mental confusion
- Dizziness
- Narrow pulse pressure


'Wet and Warm' patient (typically elevated or normal systolic blood pressure)
Vascular type – fluid redistribution Hypertension predominates
Cardiac type – fluid accumulation Congestion predominates
• Vasodilator
• Diuretic
• Diuretic
• Vasodilator
• Ultrafiltration (consider if diuretic resistance)

Decongestion Strategy

- IV loop diuretics
  - Institute EARLY in the ER
  - Dose should equal or exceed PO dose
  - Furosemide PO to IV conversion 2:1
  - Furosemide 40 mg = Torsemide 10 mg

- To enhance diuretic effectiveness
  - AC PO dose
  - Limit sodium intake (?)

HFSA 2010 Practice Guideline

Acute HF—Sodium

Recommendation 12.12

A low sodium diet (2 g daily) is recommended for most hospitalized patients.

Strength of Evidence = C

In patients with recurrent or refractory volume overload, stricter sodium restriction may be considered.

Strength of Evidence = C

Heart Failure Society of America

Lindenfeld J, et al. HFSA 2010 Comprehensive Heart Failure Guideline. J Card Fail 2010;16:e1-e194.

HFSA 2010 Practice Guideline

Acute HF—Fluid Restriction

Recommendation 12.13

Fluid restriction (<2 liters/day):

- **Is recommended in patients with moderate hyponatremia (serum sodium < 130 mEq/L)**
- **Should be considered to assist in treatment of fluid overload in other patients.** *Strength of Evidence = C*

In patients with severe (serum sodium < 125 mEq/L) or worsening hyponatremia, stricter fluid restriction may be considered.

Strength of Evidence = C

Heart Failure Society of America

Lindenfeld J, et al. HFSA 2010 Comprehensive Heart Failure Guideline. J Card Fail 2010;16:e1-e194.

Order for one day	Order for continuation

IV furosemide

- ☐ If on furosemide as an outpatient
  Total daily dose
  as IV _____mg; max 180 mg

No po furosemide at home

- ☐ Cr < 2.0 : Start with 40 mg IVP
- ☐ Cr > 2.0 : Start with 80 mg IVP

Goal :
UOP > 250-500 mL in 2 hours

Inadequate response
double previous IV dose (max = 360 mg)

- ☐ Low salt diet (Na < 2 g/day)
- ☐ Fluid restriction (2000 cc/24h)
  if Na <125 mg/dL restrict
  fluid to 1500 cc/24hr

In administration of loop diuretics which statement is correct ?

A. Bolus dosing results in less diuresis and less clinical improvement than continuous infusion

B. Continuous infusion results in worsened renal function compared to bolus dosing

C. Higher dose of diuretic results in faster weight loss and a shorter hospital stay than a lower dose diuretics

D. None of the above

DOSE Trial

The NEW ENGLAND JOURNAL of MEDICINE

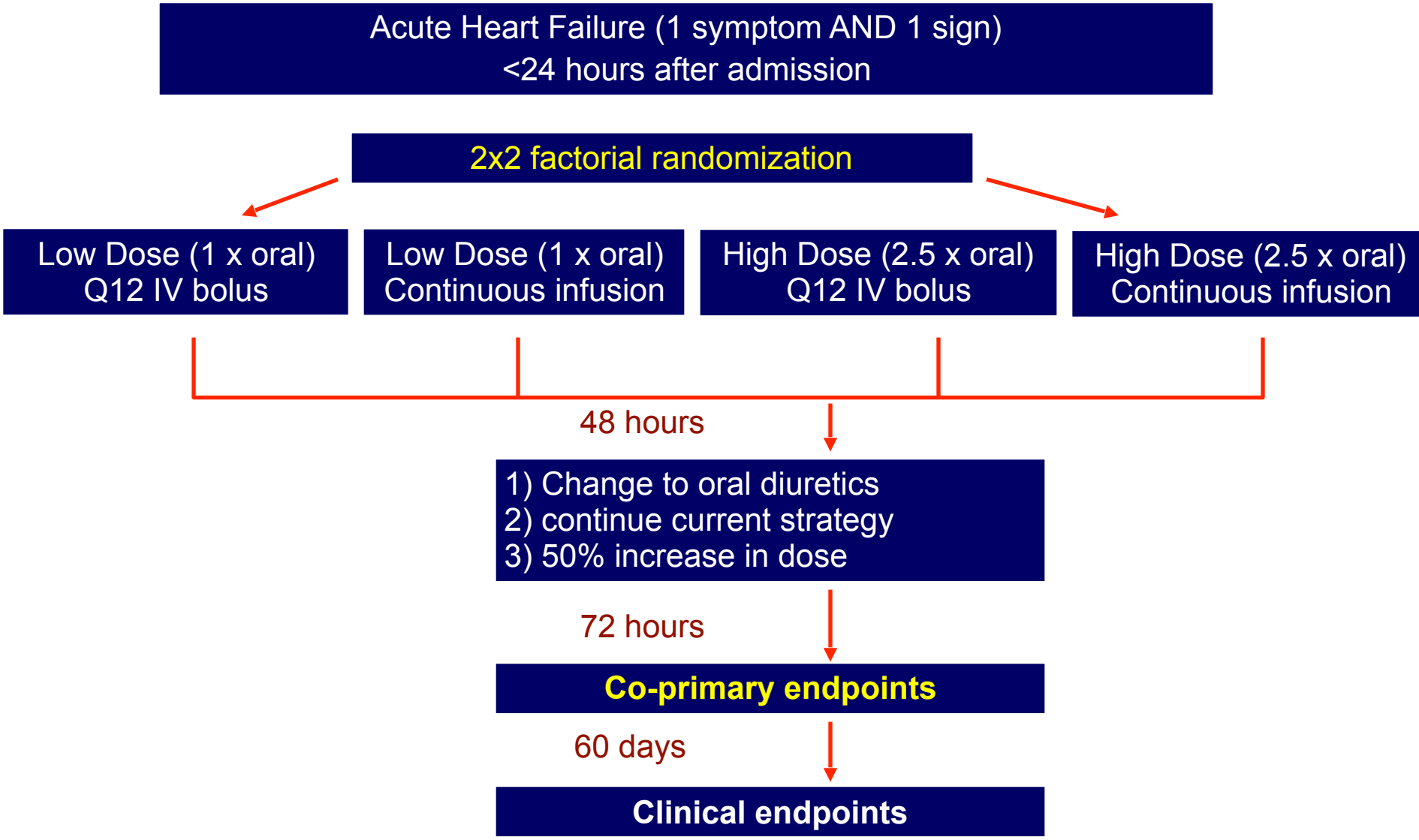
ESTABLISHED IN 1812 MARCH 3, 2011 VOL. 364 NO. 9

Diuretic Strategies in Patients with Acute Decompensated Heart Failure

G. Michael Felker, M.D., M.H.S., Kerry L. Lee, Ph.D., David A. Bull, M.D., Margaret M. Redfield, M.D., Lynne W. Stevenson, M.D., Steven R. Goldsmith, M.D., Martin M. LeWinter, M.D., Anita Deswal, M.D., M.P.H., Jean L. Rouleau, M.D., Elizabeth O. Ofili, M.D., M.P.H., Kevin J. Anstrom, Ph.D., Adrian F. Hernandez, M.D., Steven E. McNulty, M.S., Eric J. Velazquez, M.D., Abdallah G. Kfoury, M.D., Horng H. Chen, M.B., B.Ch., Michael M. Givertz, M.D., Marc J. Semigran, M.D., Bradley A. Bart, M.D., Alice M. Mascette, M.D., Eugene Braunwald, M.D., and Christopher M. O'Connor, M.D., for the NHLBI Heart Failure Clinical Research Network*

Felker et al. NEJM 2011;364:797

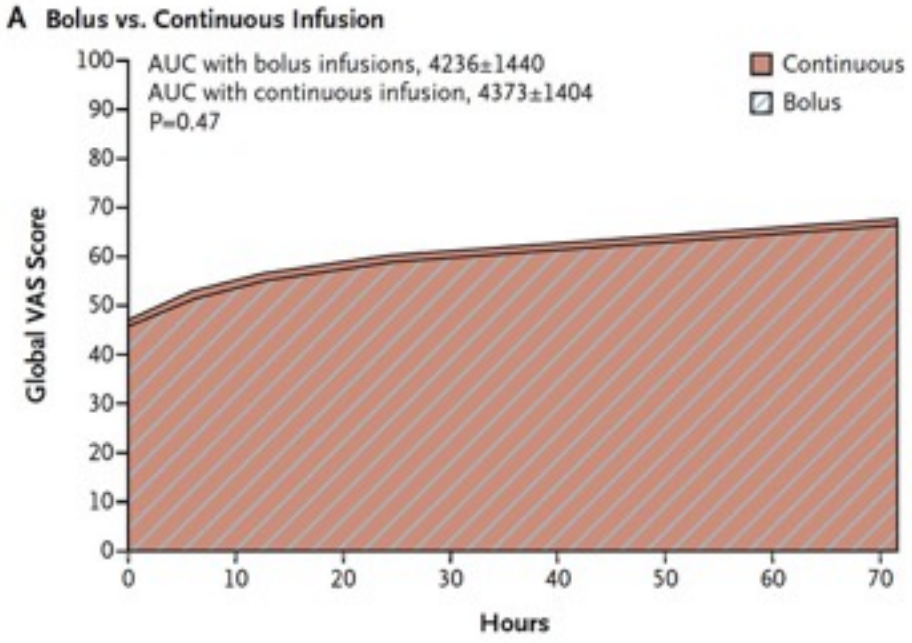
DOSE Trial : Study Design

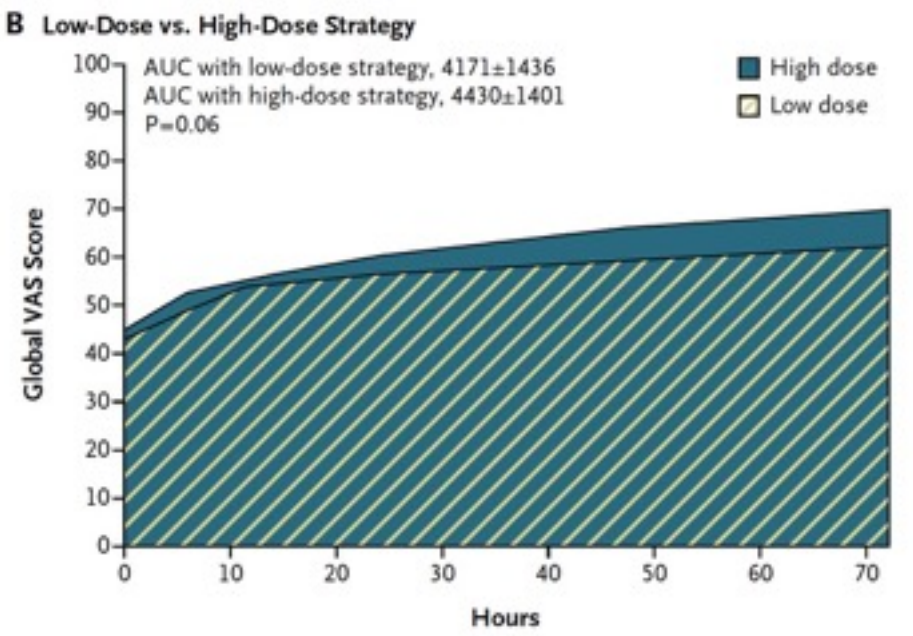


Felker et al. NEJM 2011;364:797

DOSE Study

Symptoms Relief (VAS)

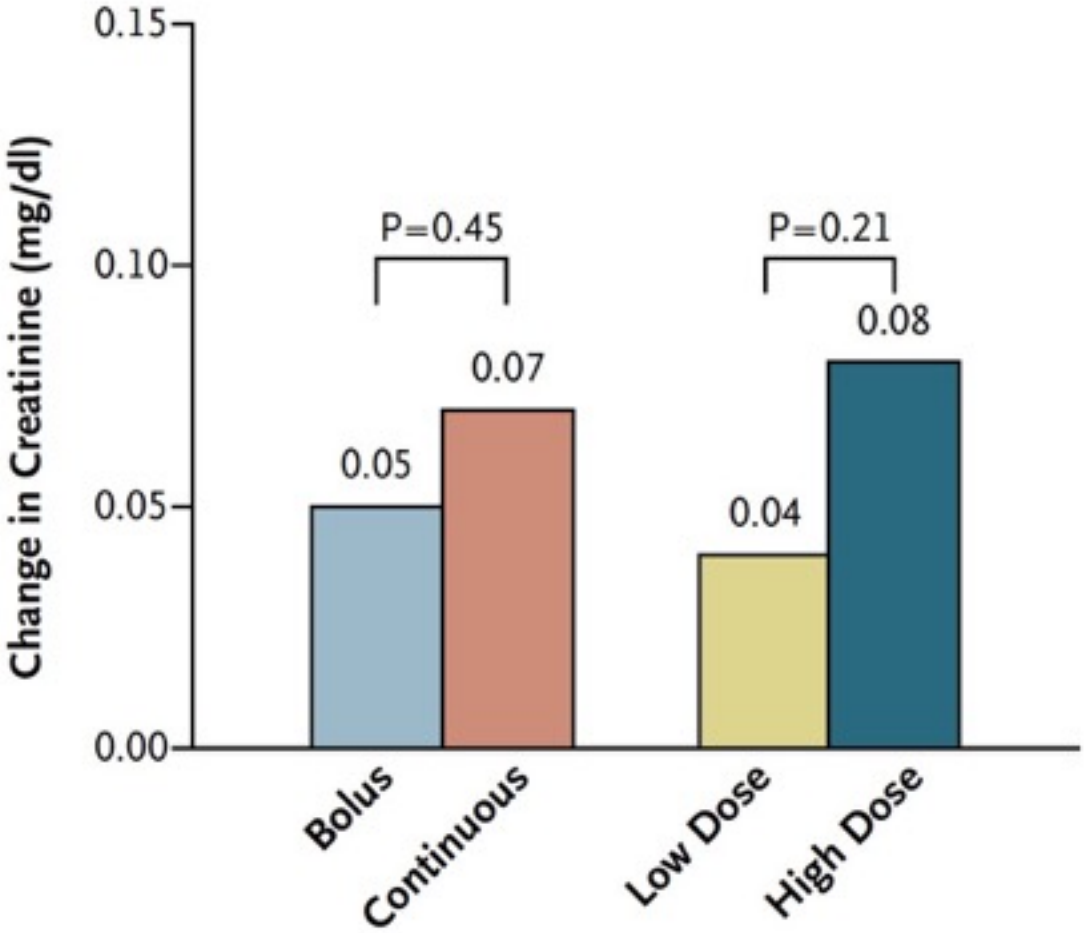




Felker et al. NEJM 2011;364:797

DOSE Study

Change in serum Cr at 72 hours



Felker et al. NEJM 2011;364:797

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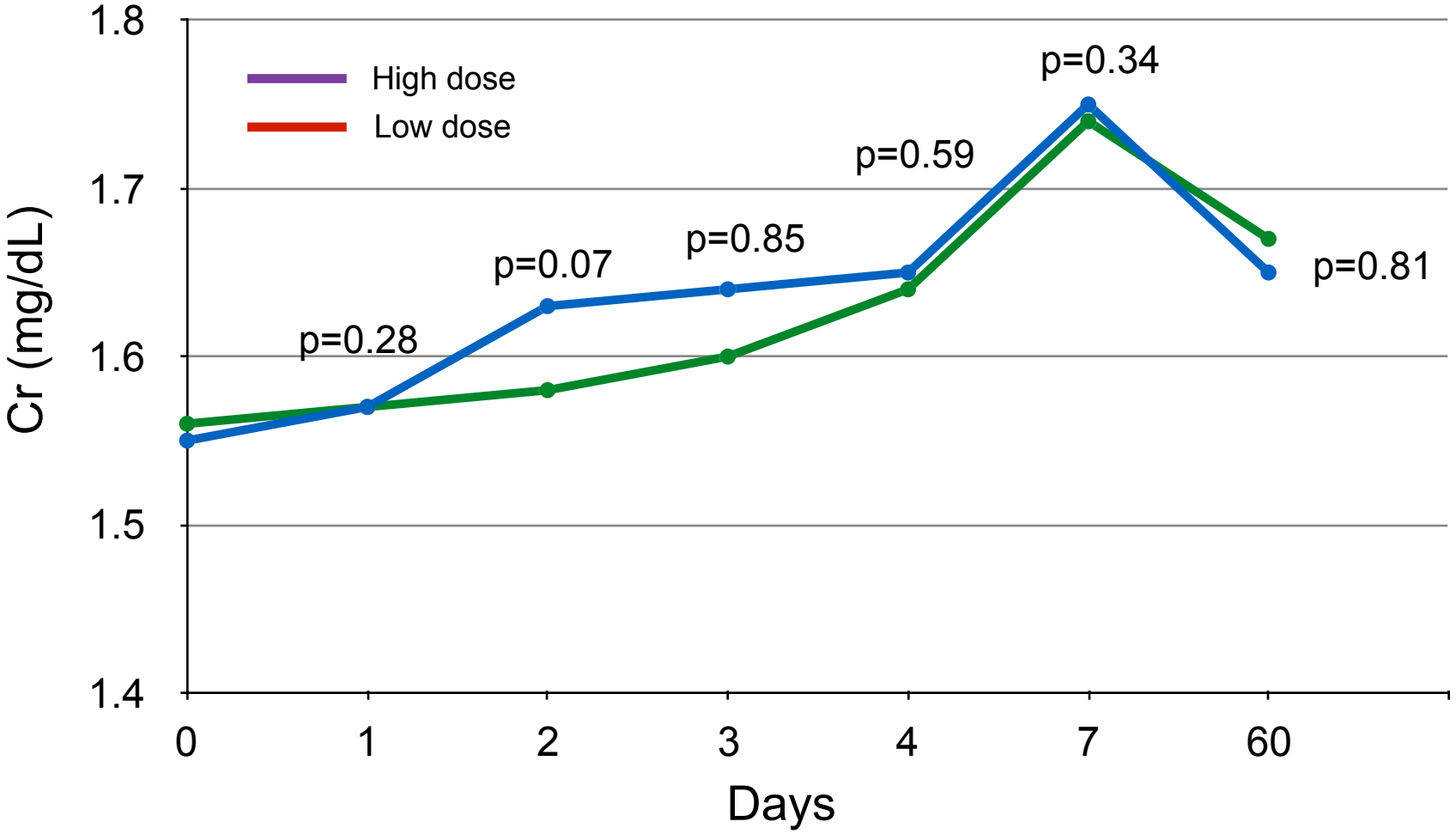

Felker et al. NEJM 2011;364:797

DOSE Study

Take Home Messages

- No substantial outcome difference between equal doses of continuous infusion Vs twice daily bolus injection of furosemide
- Higher doses may be somewhat more efficacious (2.5 x previous daily oral dose)
- Average furosemide dose used in DOSE was 100 mg q 12 hrs up to 300 mg BID x 3 days

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Intravenous Diuretic Therapy for the Management of Heart Failure and Volume Overload in a Multidisciplinary Outpatient Unit

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Craig Stevens, PharmD,* Roman M. Belenkiy, PharmD,* Laura J. Burpee, NP,‡ Michelle A. Young, NP,‡
Cynthia S. Weiffenbach, RN,‡ Jennifer A. Smallwood, MPH,‡ Lynne W. Stevenson, MD,‡ Akshay S. Desai, MD, MPH‡

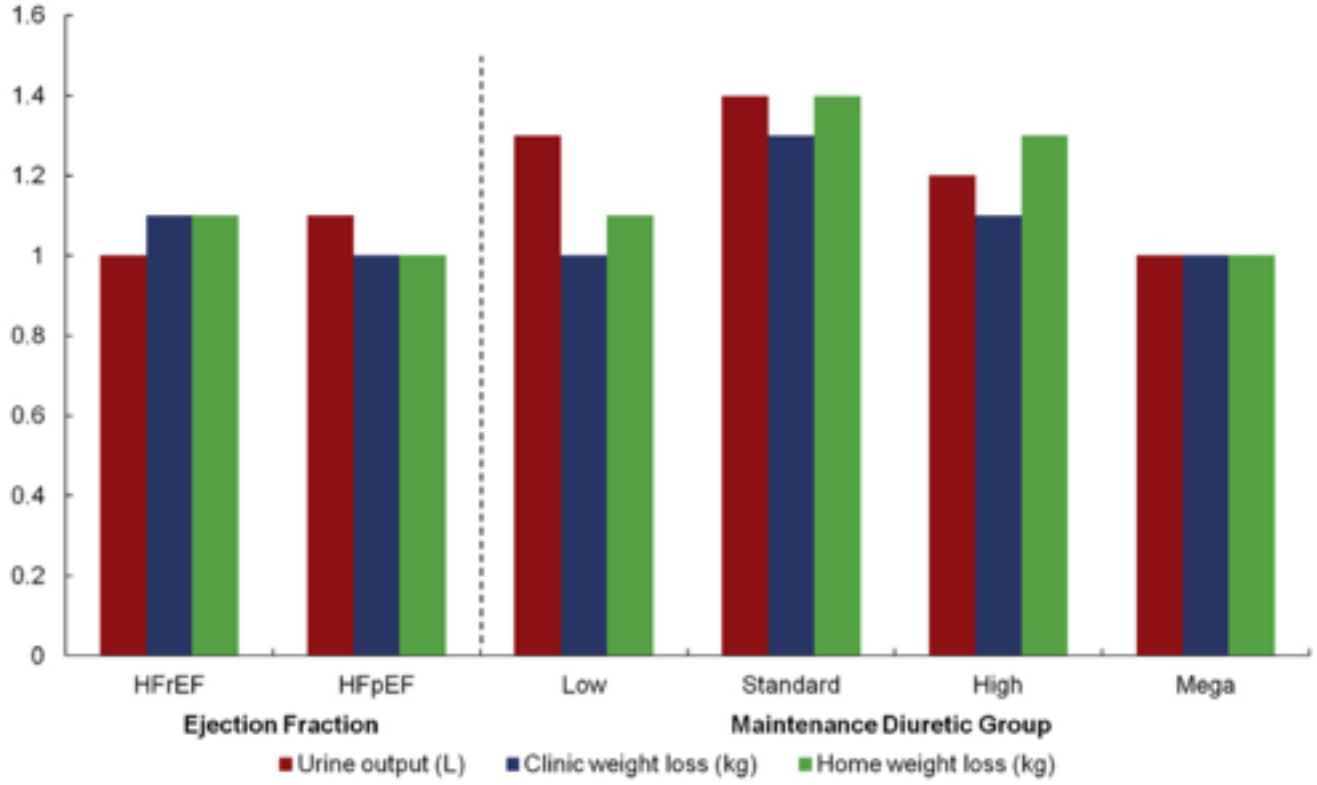
JACC Heart Failure 2016 Jan;4(1);1-8

FIGURE 1 Standardized IV Diuretic Administration Protocol

Category	Maintenance diuretic dose (mg)*	IV furosemide dose Bolus (mg)	IV furosemide dose Infusion (mg/hr)	Optional†
Low dose	≤ 40	20	20	--
Standard dose	41-160	Numeric equivalent of maintenance diuretic dose	20	--
High dose	161-300	200	20	200 mg
Mega dose	≥ 301	200	20	200 mg Thiazide diuretic‡

JACC Heart Failure 2016 Jan;4(1);1-8

FIGURE 4 Efficacy Outcomes in Notable Subgroups



Weight loss was expressed in kilograms and urine output in liters. Outcomes were similar between patients with heart failure with reduced ejection fraction (HFrEF) and heart failure with preserved ejection fraction (HFpEF). Successful decongestion was achieved in the majority of patients.

Regarding low dose dopamine in ADHF, which statement is correct ?

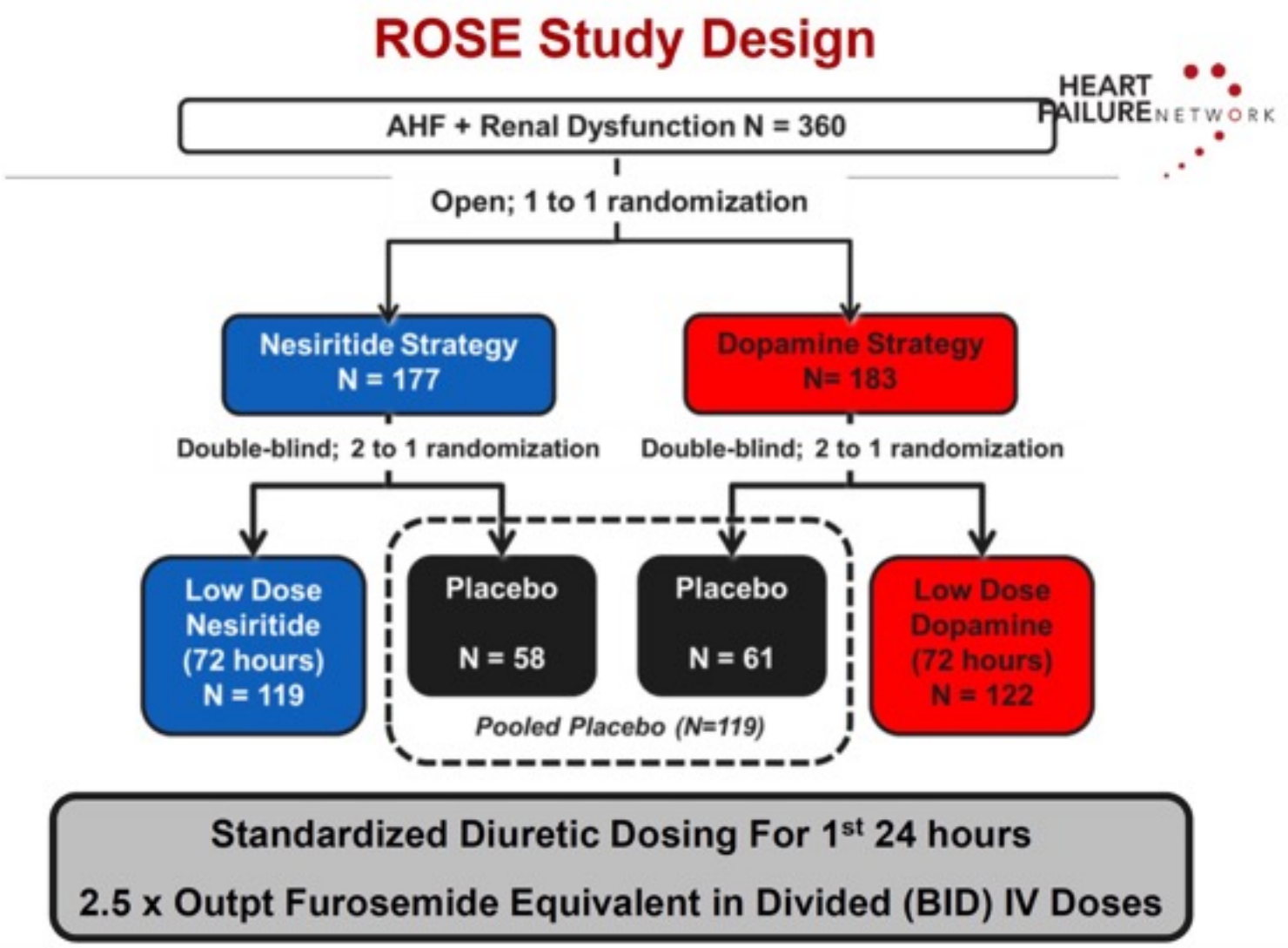
A. Low dose dopamine results in more diuresis at 72 hours when compared to placebo

B. Low dose dopamine results in more cystatin-C change when compared to placebo

C. Low dose nesiritide is better than low dose dopamine for renal outcome

D. Neither low dose nesiritide nor low dose dopamine results in more diuresis at 72 hours when compared to placebo

Low Dose Dopamine Vs Low Dose Nesiritide

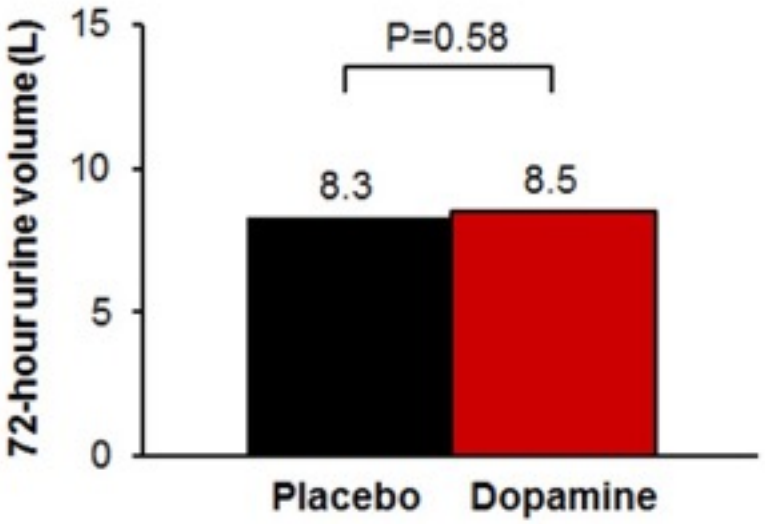


Chen HH et al. JAMA 2013

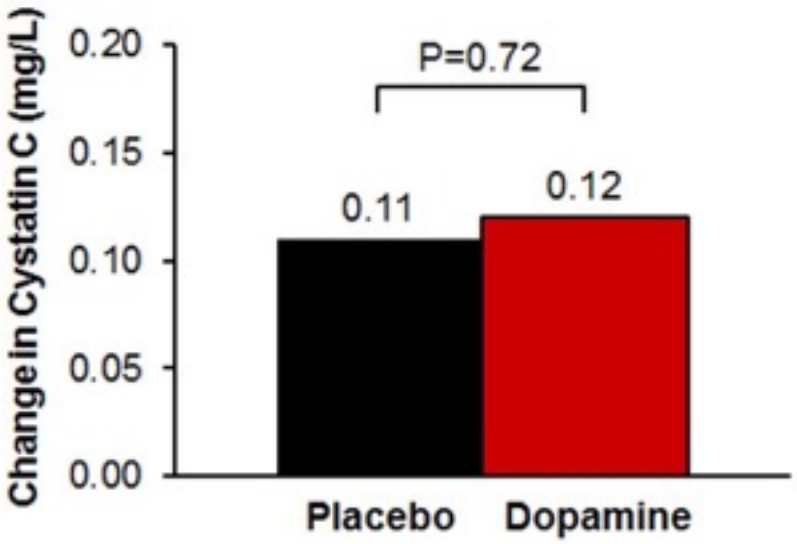
Low Dose Dopamine:
Co-primary End-points

HEART FAILURE NETWORK

72 Hour Urine Volume



Change in Cystatin-C

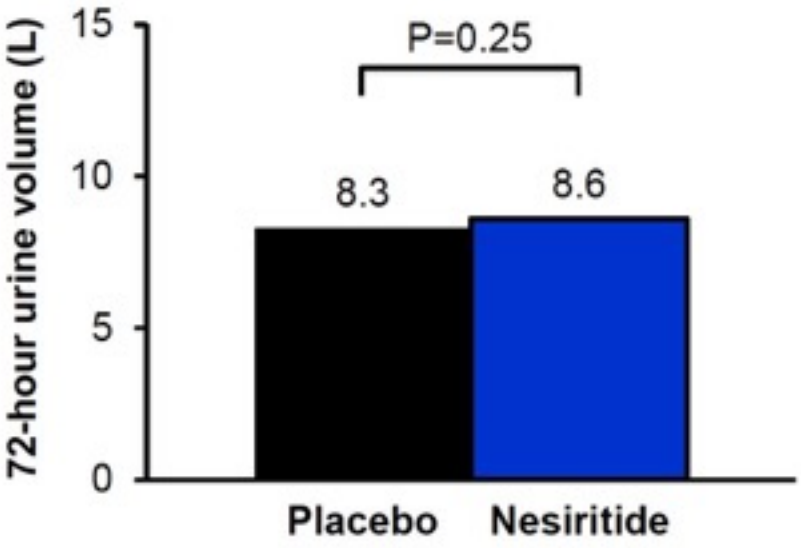


Chen HH et al. JAMA 2013

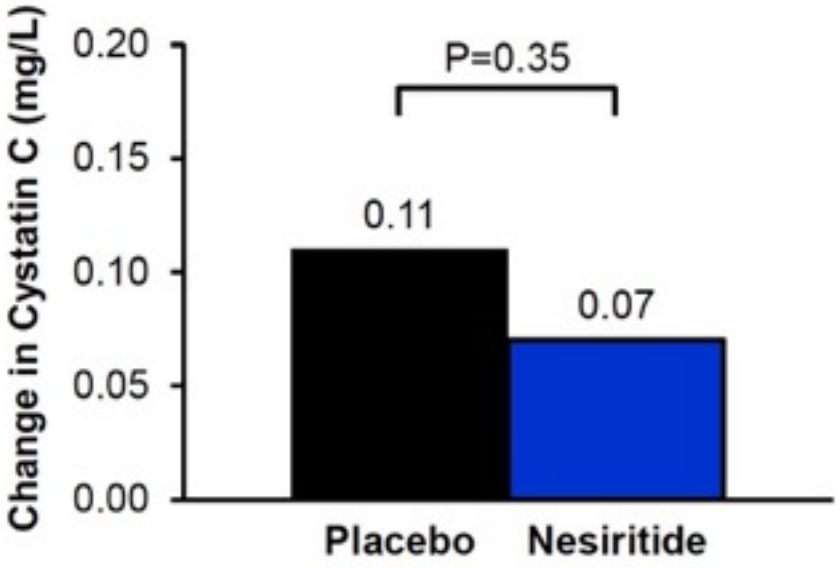
Low Dose Nesiritide
Co-primary End-points

HEART FAILURE NETWORK

72 Hour Urine Volume



Change in Cystatin-C



Chen HH et al. JAMA 2013

Safety Endpoints

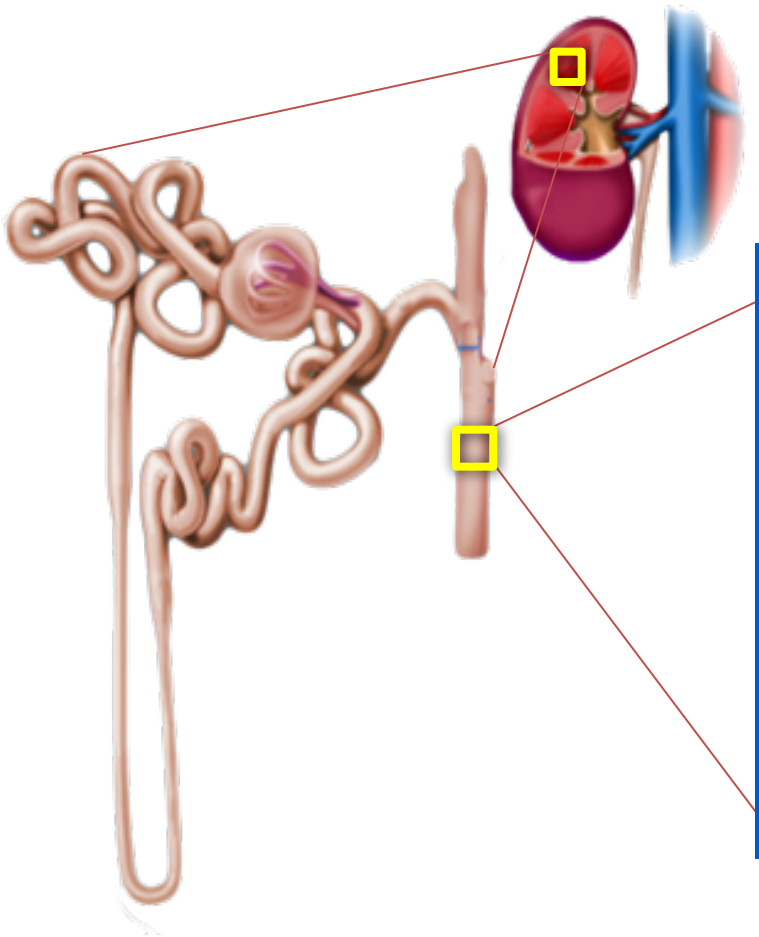
HEART FAILURE NETWORK

Study Drug Tolerance	Dopamine (n=122)	Placebo (N = 119)	P Value
Study drug d/c - Hypotension	0.9%	10.4%	<0.001
Study drug d/c - Tachycardia	7.2%	0.9%	<0.001
Study drug d/c – Any Cause	23%	25%	0.72

Study Drug Tolerance	Nesiritide (n=119)	Placebo (N = 119)	P Value
Study drug d/c - Hypotension	18.8%	10.4%	0.07
Study drug d/c - Tachycardia	0%	0.9%	0.50
Study drug d/c – Any Cause	25%	25%	0.94

Chen HH et al. JAMA 2013

Arginine Vasopressin Antagonists
Tolvaptan : Site of action



V2 Receptor : Free water absorption

Effects of Oral Tolvaptan in Patients Hospitalized for Worsening Heart Failure

The EVEREST Outcome Trial

JAMA. 2007;297:1319-1331

Marvin A. Konstam, MD
Mihai Gheorghiade, MD
John C. Burnett, Jr, MD
Liliana Grinfeld, MD
Aldo P. Maggioni, MD
Karl Swedberg, MD
James E. Udelson, MD
Faiez Zannad, MD
Thomas Cook, PhD
John Ouyang, PhD
Christopher Zimmer, MD
Cesare Orlandi, MD
for the Efficacy of Vasopressin Antagonism in Heart Failure Outcome Study With Tolvaptan (EVEREST) Investigators

Context Vasopressin mediates fluid retention in heart failure. Tolvaptan, a vasopressin V_2 receptor blocker, shows promise for management of heart failure.

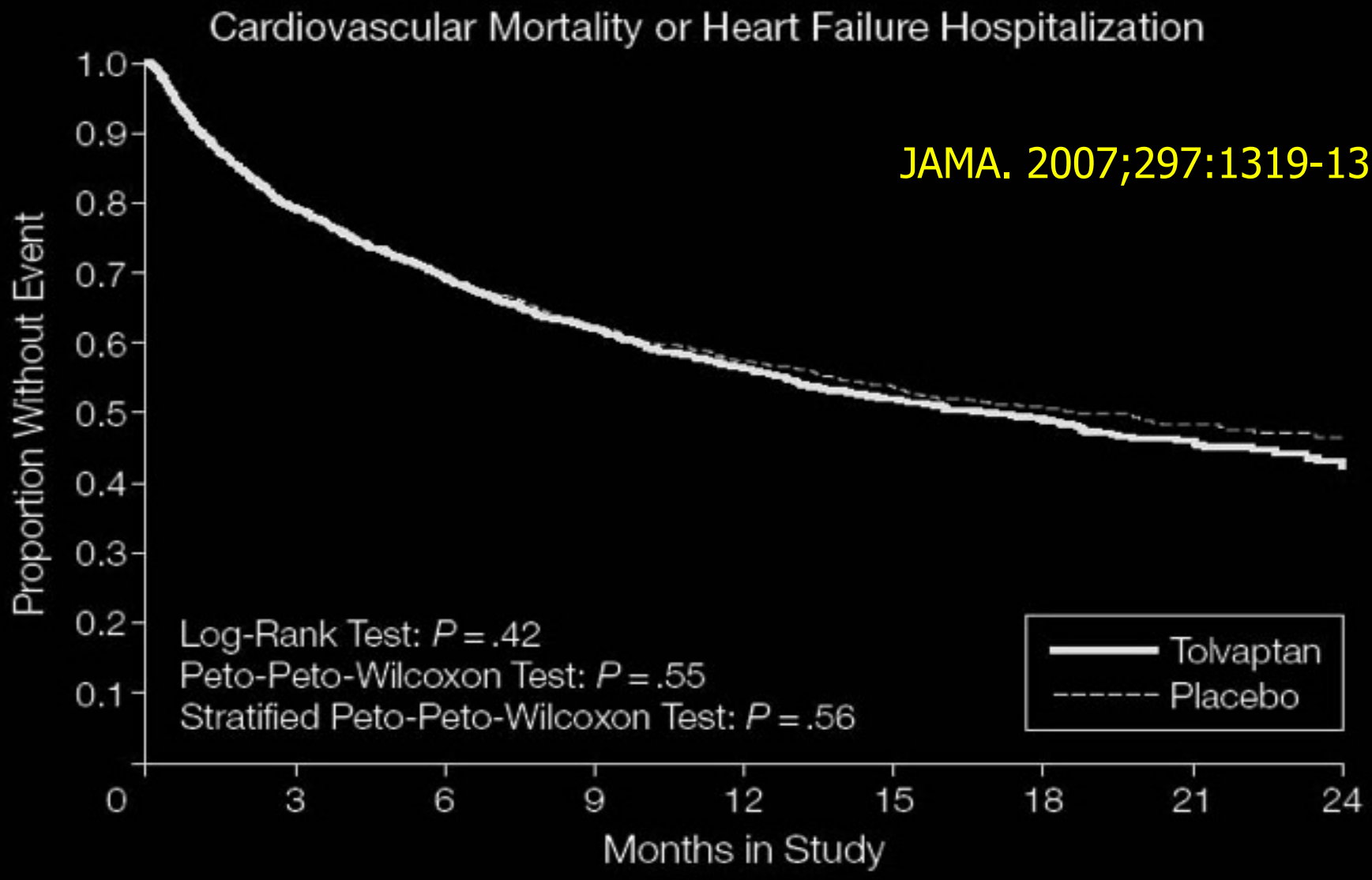
Objective To investigate the effects of tolvaptan initiated in patients hospitalized with heart failure.

Design, Setting, and Participants The Efficacy of Vasopressin Antagonism in Heart Failure Outcome Study With Tolvaptan (EVEREST), an event-driven, randomized, double-blind, placebo-controlled study. The outcome trial comprised 4133 patients within 2 short-term clinical status studies, who were hospitalized with heart failure, randomized at 359 North American, South American, and European sites between October 7, 2003, and February 3, 2006, and followed up during long-term treatment.

Intervention Within 48 hours of admission, patients were randomly assigned to receive oral tolvaptan, 30 mg once per day (n=2072), or placebo (n=2061) for a minimum of 60 days, in addition to standard therapy.

Main Outcome Measures Dual primary end points were all-cause mortality (superiority and noninferiority) and cardiovascular death or hospitalization for heart failure (superiority only). Secondary end points included changes in dyspnea, body weight, and edema.

Results During a median follow-up of 9.9 months, 537 patients (25.9%) in the tolvaptan group and 543 (26.3%) in the placebo group died (hazard ratio, 0.98; 95% confidence interval [CI], 0.87-1.11; *P*=.68). The upper confidence limit for the mortality difference was within the prespecified noninferiority margin of 1.25 (*P*< .001). The composite


Cardiovascular Mortality or Heart Failure Hospitalization
JAMA. 2007;297:1319-1331
Proportion Without Event
1.0
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
Log-Rank Test: P = .42
Peto-Peto-Wilcoxon Test: P = .55
Stratified Peto-Peto-Wilcoxon Test: P = .56
Tolvaptan
Placebo
0
3
6
9
12
15
18
21
24
Months in Study
2072 1562 1446 834 607 396 271 149 58
2061 1532 1137 819 597 385 255 143 55

EVEREST : Key Entry Criteria

Inclusions

- Hospitalized for decompensated HF <48 hours
- LVEF ≤ 40%
- Fluid overload; >2 of the following :
  - Jugular venous distention
  - Pitting edema (>1+)
  - Dyspnea

Exclusion

- Recent of planned revascularization or device implant
- STEMI during hospitalization
- SBP < 90 mmHg
- Cr > 3.5 mg%, K > 5.5 mEq/L; Hb <9%

Konstam MA *et al*. JAMA. 2007;297(12):1319-1331.

EVEREST : Conclusions

- In pts hospitalized with HF, oral tolvaptan 30 mg OD, facilitates management of volume overload with
  - Early and sustained weight reduction
  - Improvement in dyspnea (d1) and edema (d7)
  - Normalization of serum Na in hyponatremic pts
  - No worsening renal function
- Long-term treatment had no effect on long-term mortality or HF morbidity

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Clinical Investigations

Clinical Course of Patients With Hyponatremia and Decompensated Systolic Heart Failure and the Effect of Vasopressin Receptor Antagonism With Tolvaptan

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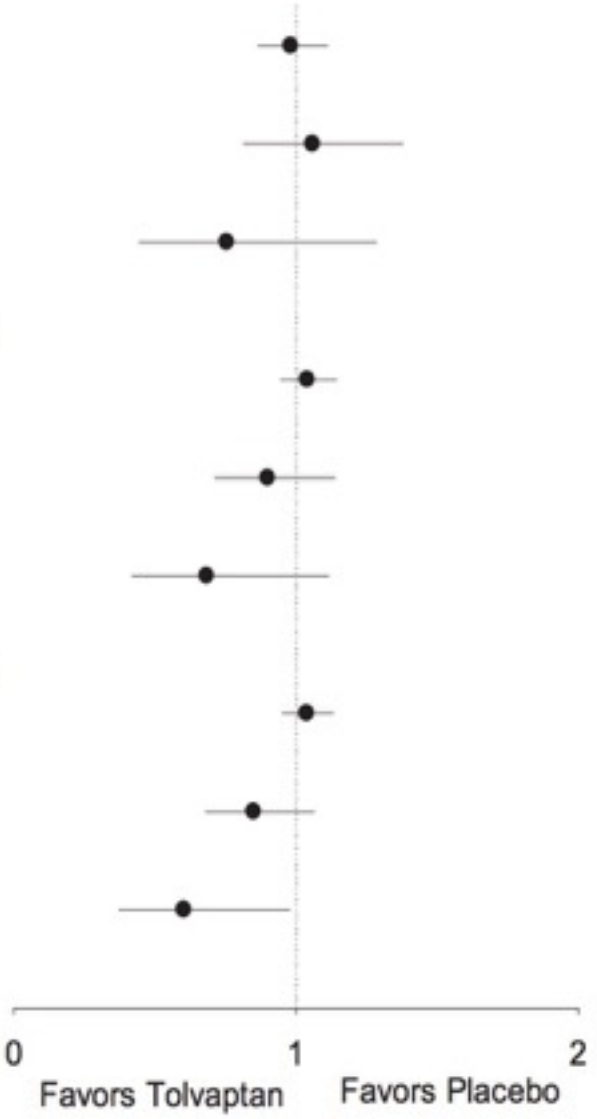
St. Louis, Missouri; Rochester, Minnesota; Chicago, Illinois; Buenos Aires, Argentina; Boston, Massachusetts; Rockville, Maryland; Florence, Italy; Gothenburg, Sweden; Nancy, France

ABSTRACT

Background: Patients with decompensated heart failure, volume overload, and hyponatremia are challenging to manage. Relatively little has been documented regarding the clinical course of these patients during standard in-hospital management or with vasopressin antagonism.

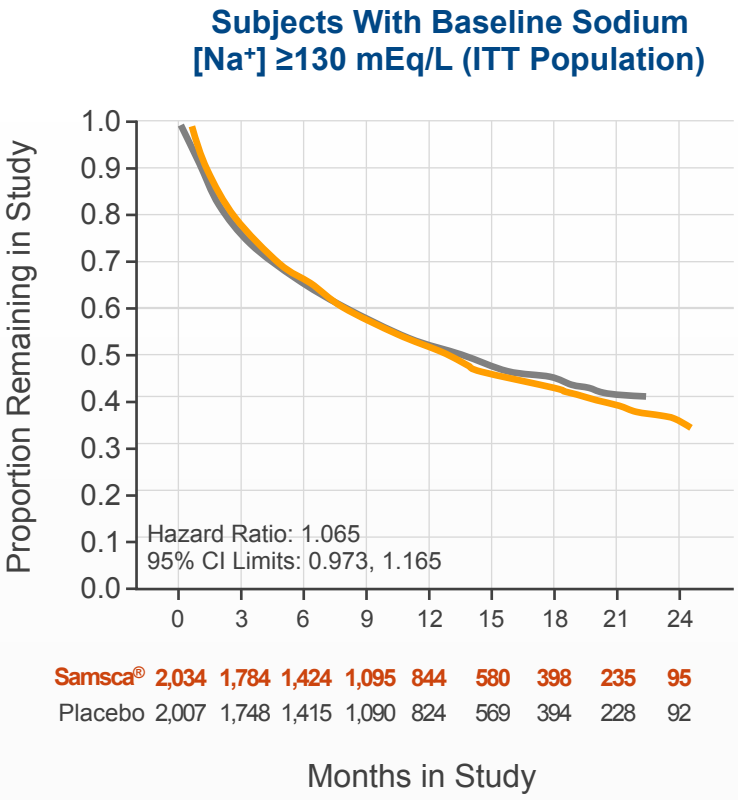
Methods and Results: The Efficacy of Vasopressin Antagonism in Heart Failure Outcome Study With Tolvaptan database was examined to assess the short-term clinical course of patients hospitalized with heart failure and hyponatremia and the effect of tolvaptan on outcomes. In the placebo group, patients

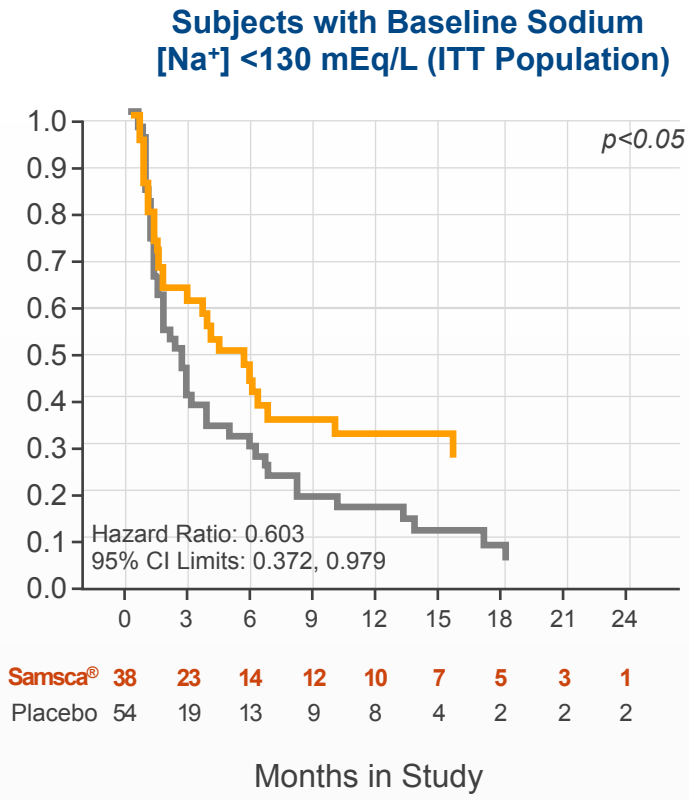
Time to All-cause Mortality		# Subjects	# Events	P-value
EVEREST All Patients	TLV	2072	537	0.76
	PLC	2061	543	
Na+ < 135 mEq/L	TLV	243	116	0.67
	PLC	232	106	
Na+ < 130 mEq/L	TLV	38	22	0.30
	PLC	54	36	
Time to First Occurence of CV Mortality or HF Hospitalization				
EVEREST All Patients	TLV	2072	871	0.42
	PLC	2061	829	
Na+ < 135 mEq/L	TLV	243	137	0.38
	PLC	232	142	
Na+ < 130 mEq/L	TLV	38	26	0.12
	PLC	54	43	
Time to First Occurence of CV Mortality or CV Hospitalization				
EVEREST All Patients	TLV	2072	1006	0.37
	PLC	2061	958	
Na+ < 135 mEq/L	TLV	243	146	0.16
	PLC	232	158	
Na+ < 130 mEq/L	TLV	38	26	0.04
	PLC	54	46	



Hauptman J J Cardiac Fail 2013;19:390e397

Patients with Heart Failure and Hyponatremia





Overall CV Mortality/Morbidity (ITT) HR 1.04; 95%CI (.95-1.14).

Placebo tolvaptan

Arginine Vasopressin Antagonists

In patients hospitalized with volume overload, including HF, who have persistent severe hyponatremia and are at risk for or having active cognitive symptoms despite water restriction and maximization of GDMT, vasopressin antagonists may be considered in the short term to improve serum sodium concentration in hypervolemic, hyponatremic states with either a V2 receptor selective or a nonselective vasopressin antagonist.

Practical Use of Tolvaptan

- Start in-hospital, start dose 7.5/15 mg, maximum dose at 60 mg OD
- Frequent monitoring of serum [Na^+] (at least q 8 hr on D1 and daily onward)
- Stop all fluid restriction (especially first 24 hours of therapy)

Ultrafiltration

Ultrafiltration may be considered for patients with refractory congestion, who failed to respond to diuretic-based strategies

CARRESS-HF, UNLOAD

Renal replacement therapy should be considered in patients with refractory volume overload and acute kidney injury

K > 6.5 mEq/L, pH <7.2, BUN> 125 mg/dL, Cr > 3.4 mg/dL

Summary

When congestion fails to improve in response to diuretics, consider

1. Reevaluate presence/absence of congestion
2. Sodium/fluid restriction
3. Increasing dose of loop diuretics
4. Continuous IV infusion diuretics
5. Sequential nephron blockade
6. Optimize hemodynamics (PAC-guided therapy)
7. Vasopressin antagonists
8. Ultrafiltration

Case #2

58-year-old male

Longstanding hypertensive heart disease, EF 60%

2 days of increasing dyspnea, orthopnea

BP 190/100, PR 64/min, warm extremities,
rales halfway up both lung fields, JVP 14 cmH2O
hypertensive retinal change

Labs : Normal CBC, Cr 1.9 (baseline 1.4)

ECG : No ischemia

What is the best initial therapy

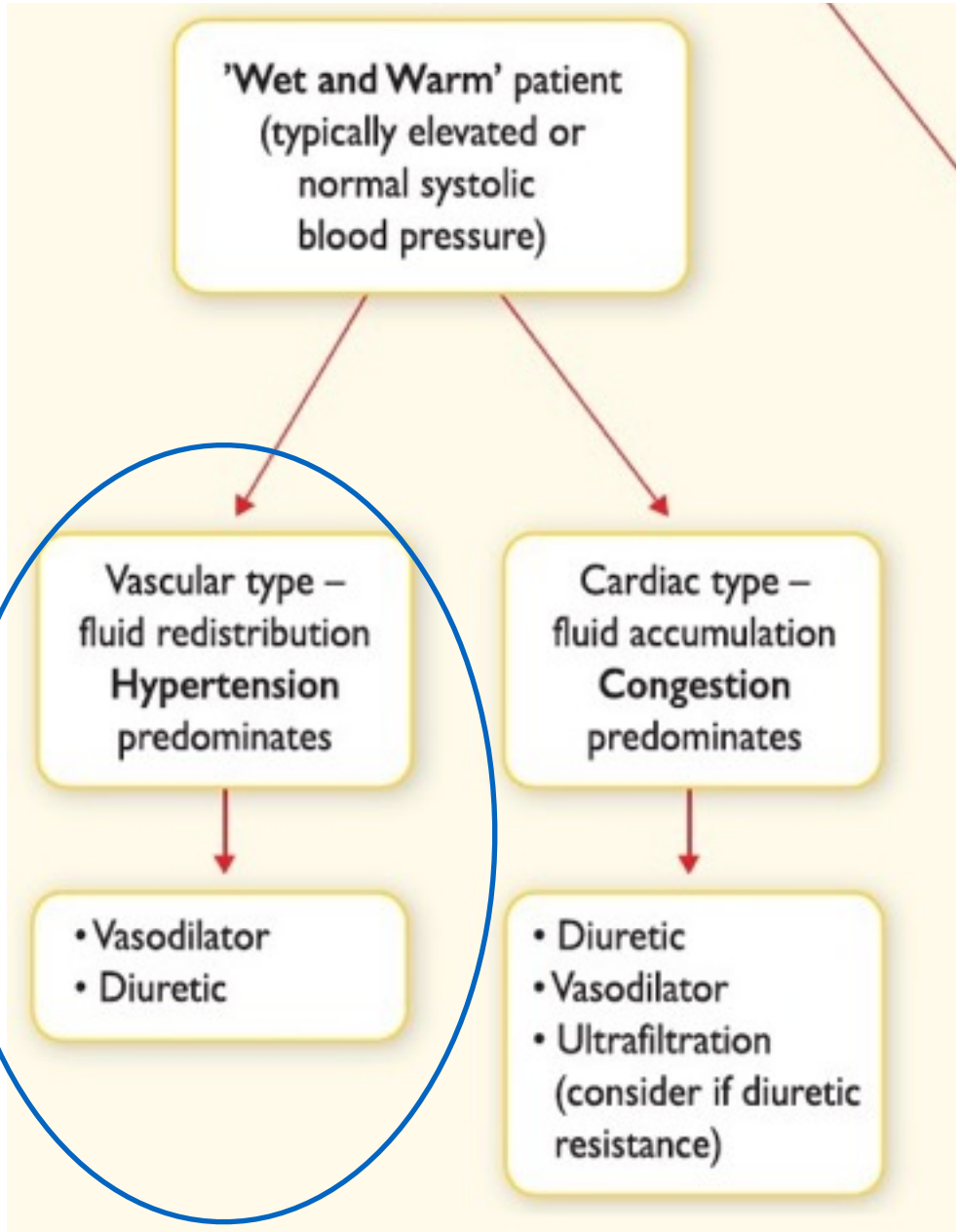
A. Milrinone drip

B. Start IV furosemide + IV NTG

C. Start IV furosemide and HCTZ

D. Add Hydrazine and ISDN

E. Add Lisinopril and amlodipine, follow BP's


'Wet and Warm' patient (typically elevated or normal systolic blood pressure)
Vascular type – fluid redistribution Hypertension predominates
Cardiac type – fluid accumulation Congestion predominates
• Vasodilator
• Diuretic
• Diuretic
• Vasodilator
• Ultrafiltration (consider if diuretic resistance)

IV Vasodilators : Overview

- In acute HF associated with
  1. Acute mitral regurgitation
  2. Acute aortic regurgitation
  3. Severe hypertension

- Beneficial effects :
  - Decrease BP and improve the efficacy of cardiac work
  - Speed symptoms relief
  - Possibly decrease risk for CCU, mechanical ventilation
  - **No proven change in mortality**

- Nitroglycerin, Nitroprusside, Nesiritide

Nitroglycerin

- For patients with SBP > 90 mmHg (and without symptomatic hypotension)

  - Nitroglycerin 0.6 mg sublingually, repeated every 5-10 mins for 3-4 doses

  - Nitroglycerin IV

    Starting dose : 10-20 mcg/min titrate 5-10 mcg/min every 5 minutes (maximal dose 200 mcg/min)

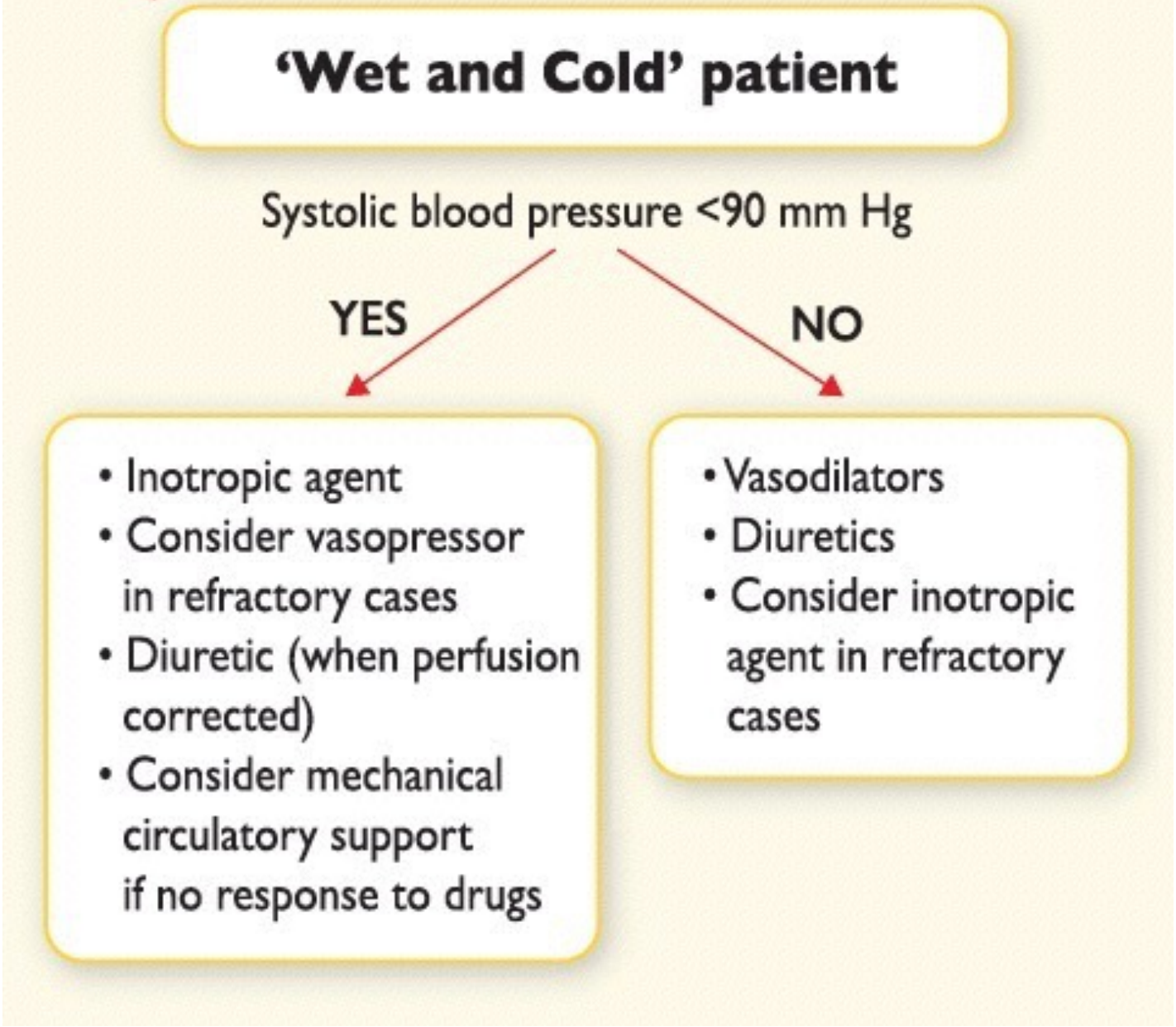
Nitroprusside

- Primary arteriolar dilator
  - Dose :
    Start at 0.3 mcg/kg/min
    Titrate upward by 0.2 mcg/kg/min at 3-5 mins interval
    Maximum dose 5 mcg/kg/min

  - Nitroprusside toxicities :
    - Cyanide intoxication : Metabolic acidosis
    - Thiocyanate toxicity : Hyperreflexia, seizures, altered mentation

Advantages
- Potent
- Fine titration

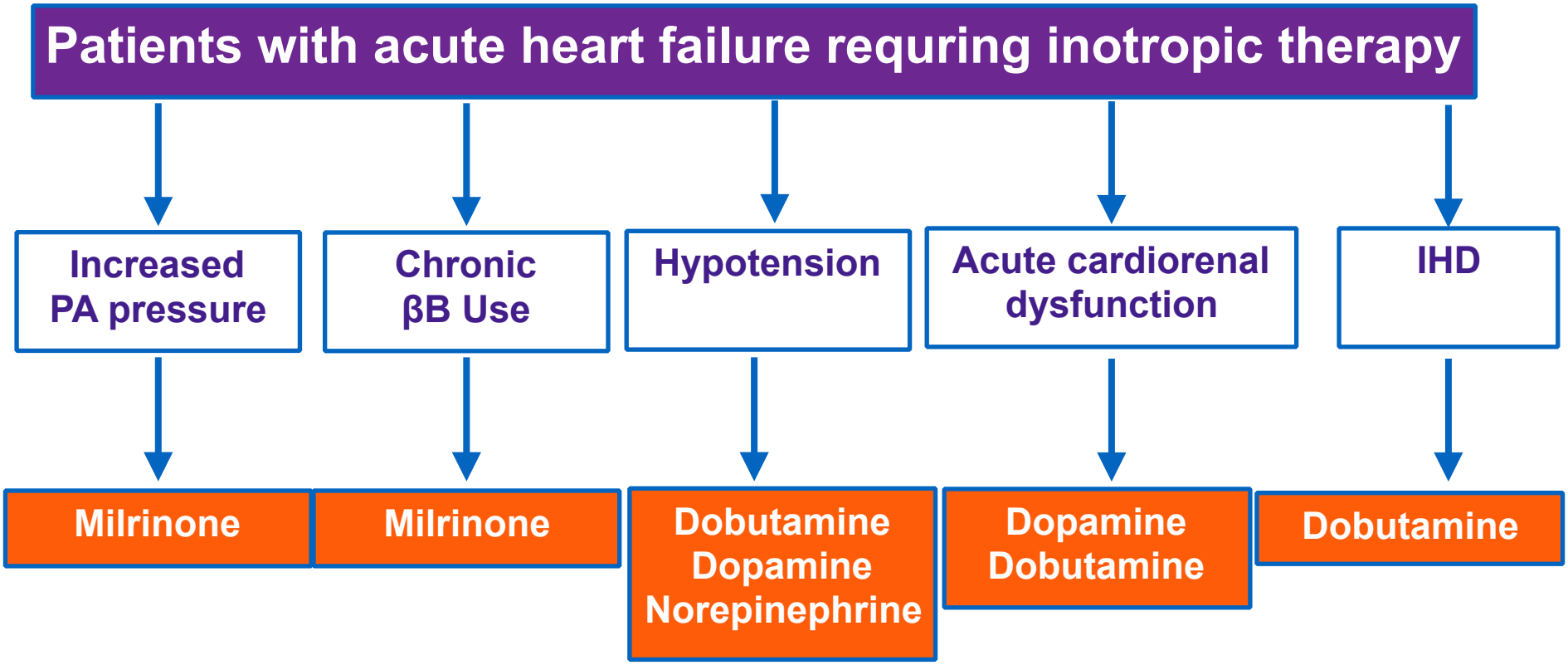
Disadvantages
- CCU and arterial line
- Thiocyanate toxicity esp in renal/hepatic insufficiency
- No randomized trials


'Wet and Cold' patient
Systolic blood pressure <90 mm Hg
YES
NO
• Inotropic agent
• Consider vasopressor in refractory cases
• Diuretic (when perfusion corrected)
• Consider mechanical circulatory support if no response to drugs
• Vasodilators
• Diuretics
• Consider inotropic agent in refractory cases

Properties of Beta-stimulants, Inotropic vasodilators (inodilators)

	α>β	β1 stimulation	Mixed β1 & β2 effects	PDE inhibitors	Dopaminergic
Drug example	NE	Dobutamine (also some β2)	Epinephrine (also some alpha)	Milrinone	Dopamine
Inotropic effects	++	++	+++	+	++
Arteriolar vasodilation	0	+	+	++	+
Vasoconstriction	+++	0	++	0	+
Chronotropic effect	+	+	++	+	+
Increase in BP	+++	0/+ (by ↑CO)	++	-	0/+ (vasocons)
Use in CHF	+	++	0	++	++

Selecting the proper inotropes



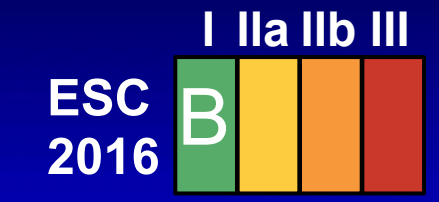
Crit Care Med 2008; 36: 106
Curr Opin Crit Care 2010;16:432

Positive Inotropes and Vasopressors in Acute Heart Failure

	Bolus	Infusion rate
Dobutamine	No	2-20 mcg/kg/min (β+)
Dopamine	No	<3 mcg/kg/min : Renal effect (δ+) 3-5 mcg/kg/min : Inotropic (β+) > 5 mcg/kg/min : (β+), vasopressor (α+)
Milrinone	25-75 mcg/kg over 10-20 mins	0.375-0.75 mcg/kg/min
Norepinephrine	No	0.2-1.0 mcg/kg/min
Epinephrine	Bolus 1 mg can be given IV during resuscitation repeated q 3-5 mins	0.05-0.5 mcg/kg/min

HFSA 2010 Practice Guideline

Acute HF—VT Prophylaxis



Recommendation 12.16 (NEW in 2010) 1 of 2

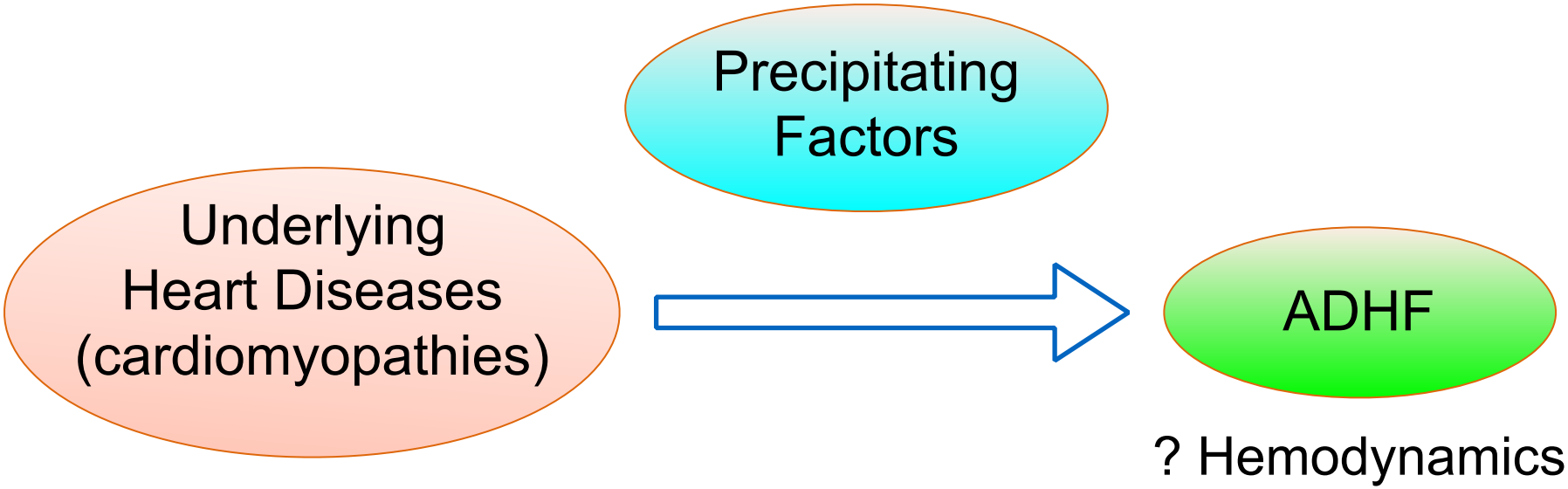
Venous thromboembolism prophylaxis with low dose unfractionated heparin, low molecular weight heparin, or fondaparinux to prevent proximal deep venous thrombosis and pulmonary embolism is recommended for patients who are admitted to the hospital with ADHF and who are not already anticoagulated and have no contraindication to anticoagulation.

Strength of Evidence = B

Heart Failure Society of America

Lindenfeld J, et al. HFSA 2010 Comprehensive Heart Failure Guideline. J Card Fail 2010;16:e1-e194.

Summary


Precipitating Factors
Underlying Heart Diseases (cardiomyopathies)
ADHF
? Hemodynamics

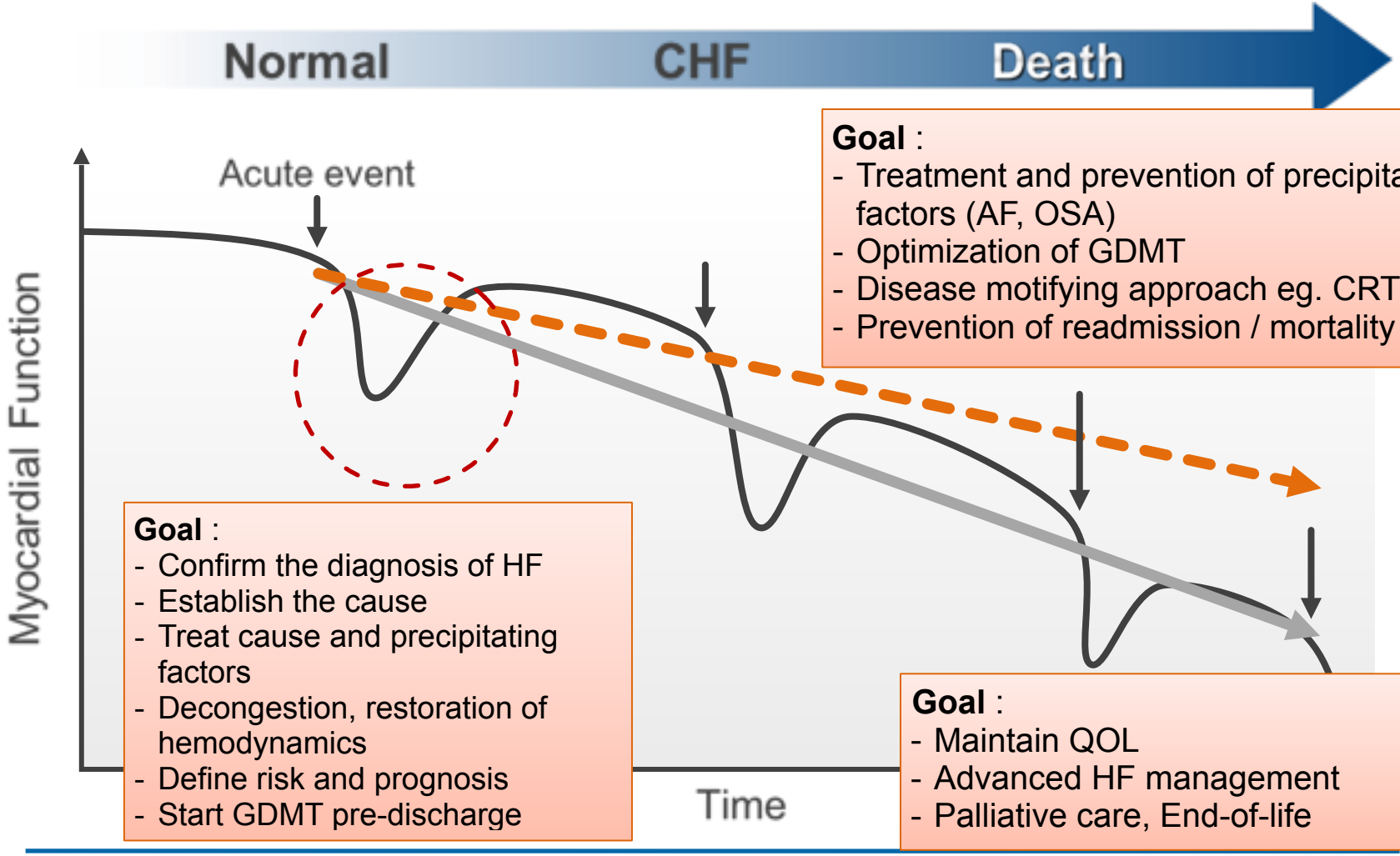
Common Precipitating factors of HF

1. Non-compliance to diet and medications
2. Myocardial ischemia
3. Poorly controlled hypertension
4. Cardiac arrhythmias (esp. AF)
5. Infections
6. Anemia
7. Worsening renal function
8. Thyroid abnormalities
9. Use of new medications (esp. NSAIDs)

Before discharging AHF patients

- Exacerbating factors addressed
- Near optimal volume status achieved
- Optimal pharmacologic therapy (ACE inhibitor/ARB and β-blocker) achieved or intolerance documented
- Comorbidities well managed
- Left ventricular ejection fraction documented
- Smoking cessation counseling initiated
- Patient and family education provided
- Follow-up visit scheduled within 7 to 10 days

Natural History of Heart Failure



Gheorghiade M. et al. Am J Cardiol 2005;96(6A):11G-17G


Heart Failure Essentials for Cardiology Fellows 2016

Thank you for your attention

Feel free to ask questions at

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